PRACTICAL CARDIOLOGY

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PREFACE

The physician today is confronted with an unprecedented challenge in the practice of medicine; a remarkably enlightened public demands and expects to receive a type of medical care and service that is based on the accepted concept that every general practitioner is an expert in the common diseases and with special emphasis upon the diagnosis and the treatment of the cardiovascular disabilities. In the agonizing reappraisal of this changing situation, the doctor recognizes the need for constant and continuing pursuit of knowledge. Since World War II, there have appeared a large number of books devoted entirely to heart disease; most of these are thick, heavy volumes and all are generally good presentations of the various aspects of the cardiovascular syndromes.

The present volume is written with no apology for it is not to be considered a complete treatise on modern cardiology and just another publication to be added to the reference shelf; rather it is a practical book concerned, as its name implies, with practical cardiology. I believe that there is a definite place for a not overwhelming technical volume in the diagnosis and treatment of most cardiovascular disabilities which may be of service to the busy general practitioner.

The book is largely based upon experiences in the practice of cardiology for the past 40 years; during this period I have seen cardiology develop from a relatively minor aspect of general medicine to a great specialty embracing wide fields of scientific medicine and surgery as well as

the technical application of devices made possible by an electronic and atomic age.

In a humble way and, it is hoped in the best traditions of medicine, this volume is written; it is based upon a life time experience in practical cardiology. It is my most sincere wish that the reader may profit in some small manner and that in his hour of need the practitioner may have *his burdens of life saving responsibilities lightened.*

A final word of thanks and gratitude to my many associates and students who over the years have made the teaching of cardiology such a rewarding experience; without their continued interest and assistance, it is doubtful that I would have been able to carry on the vast research programs in clinical cardiology which have occupied much of my time since 1926. The inspiration which I received from them has always been greater than my capacity to give instruction in cardiology in a manner worthy of their appreciation. I am indebted to the Cardiovascular Research Committee of the American College of Sports Medicine for the privilege of presenting photographs of the cardiomoulage method of demonstrating functional capacity of the heart and to the Valley Forge Heart Institute and Research Center for the utilization of certain clinical data. With a deep sense of appreciation I acknowledge the assistance of Mr. Elliot A. Daitz whose generous contributions have made the work of the Daitz Fund for Cardiovascular Research possible. To my publisher, Mr. E. K. Georg Landsberger, also a word of thanks for the opportunity of presenting a view of practical cardiology with no limitations of scope and personal opinion.

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PRACTICAL CARDIOLOGY

CHAPTER I

SOME GENERAL OBSERVATIONS

Over the keystone arch of the marble doorway, leading to the old Research Laboratory of the Witkin Foundation for the Study and Prevention of Heart Disease, the following inscription was carved:

Lucky Is The Man
For Whom The Bell Tinkles
Before It Tolls

It was placed there at the suggestion of Dr. Harlow Brooks, who, at the time in 1926, was Emeritus Professor of Medicine at New York University; it crystallized the well known clinical observation that those patients who had experienced the painful disabilities of the failing heart over a period of time, had been forewarned, and thus had an opportunity of preparing for whatever might occur. From this point of view, they were definitely *more* fortunate than the hapless individual *unexpectedly* stricken with a fatal heart attack.

This, in short, is the basic keynote of every public health agency; the programs concerned with periodic physical examinations have been focused upon the prevention of heart disease, cancer, and the infectious disease. With deaths from cardiovascular disease leading the list, it is small wonder that the public at large has become

likely to cause the individual to seek medical care but, even here, a process of rationalization frequently delays treatment; heart burn, indigestion, neuritic pains, are but a few of the self-diagnoses which are made. The appearance of an acute cardiac emergency, however, always serves as a grim indication that the initial phase of the disease process is over.

Chronic Disturbances of the Heart

In contrast to the dramatic manifestations of the acute cardiac emergency, where the factors involving life or death are completed within a few moments or hours, chronic heart disease may extend over a long period of time, sometimes for 20 to 30 years or longer. Mr. W. was a man of 56 when his physician turned him over to the author for treatment; his doctor had seen him for about 24 years. Mr. W. was a slow atrial fibrillator with semi-annual bouts of acute congestive failure. He died in such an attack at the age of 88, the sole survivor of a rather large family all of whom had been greatly concerned about his welfare. He can be considered as having suffered from "serious heart disease" for more than 56 years.

Patients with valvular pathology have a longer life expectancy now than in any previous generation; reduction in subsequent infections by antibiotics, new drugs, and a surgical approach to some valve lesions have greatly expanded the period of useful years. The hypertensive syndromes have also responded to improved therapeutic measures so that, while the actual incidence of cerebral

more and more aware of the seriousness of symptoms arising from, or associated with, disabilities of the heart and blood vessel system. It also explains, in some measure, why the cardiac emergencies lead all other types of medical conditions for which the general practitioner is called upon to treat. From the experience of the past decade it is not unreasonable to expect that such emergency calls will tend to increase; the physician must be prepared for a larger share in the care of such patients.

Acute Disturbances of the Heart

Fortunately, most acute disturbances of the heart are easily recognized, and fortunately also, most such conditions respond promptly to appropriate treatment. Some cardiovascular crises are more complicated and, while simple to diagnose, may require heroic measures provided only by adequate hospitalization facilities. In certain instances, a small group of cardiac emergencies remain diagnostic problems even after the scrutiny of the most scientific and advanced research institution. The acute emergencies are emphasized here since it is a well recognized fact in cardiac clinics that many patients with heart disease seek treatment only after a dramatic reminder of their disability. Procrastination is the rule in any chronic condition; in cardiovascular disease the onset and development of symptoms may be unsuspected by the patient. There is a common tendency to underestimate dyspnea on climbing stairs or the occurrence of ankle edema at the end of the day. Precordial pain is more

likely to cause the individual to seek medical care but, even here, a process of rationalization frequently delays treatment; heart burn, indigestion, neuritic pains, are but a few of the self-diagnoses which are made. The appearance of an acute cardiac emergency, however, always serves as a grim indication that the initial phase of the disease process is over.

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and kidney complications have not lessened, the postponement of these crises has permitted a longer symptom-free existence. The degenerative diseases of the cardiovascular system currently occupy the focus of world wide scientific interest; spectacular attention has been given to the arterial lesions which cause disturbances of the coronary, cerebral, and renal circulations. Coronary disease and strokes are headline news in the daily press. From the vast publications of research comes a slow but progressive understanding of the complicated life processes, concerned in the phenomena of aging, with the promise of new and better methods of managing this increasing clinical problem.

Cardiovascular Technology

The number of instruments, apparatus, gadgets, and methods which have been developed for the study and treatment of heart disease have continued to increase at an amazing rate in the past 15 years. The ingenuity of the cardiac physiologists coupled with the skill of the electronic engineers have been productive of a host of complicated devices which have done much to advance the front of scientific knowledge concerning the heart and blood vessel system in health and disease. In their *place, and under specific circumstances,* each and every one of these instruments of precision have a recognized role in cardiology and the practitioner may well profit by a visit to any one of the great heart research centers with which this nation is liberally blessed.

Such a visit is, however, not without certain psycho-

logic implications; the physician is likely to leave the research center with some misgivings in regard to his own adequacy and he may be no little shaken by his contact with the frontiers of modern day medical science. But this is not a dismal and gloomy picture, for sooner or later, there comes the final realization and recognition that the actual practice of medicine is indeed a different world from the research laboratory and that the battlefield of medicine at the bedside or upon the highway is no less important than the mastery of electronic apparatus used in the attempt to pry out the secrets of cardiac physiology and to break through the barrier of the unknown.

Observation of the Patient

In the acute cardiovascular crises, observation takes precedence over all other diagnostic methods; careful attention to the patient's position, color, respiration, consciousness, and speech, frequently reveals telltale clues at the very outset of the examination. Many acute cardiac emergencies present characteristic clinical pictures which are familiar to the clinician; the cyanosis of the face and frothy expectoration of pulmonary edema, the cold, clammy pallor of cardiogenic shock from coronary occlusion, the florid face and excitability of hypertensive encephalopathy, the stertorous and snoring respirations of hemiplegia from a cerebral accident, the apprehensive facies of paroxysmal tachycardia, and the frozen posture of severe angina pectoris, are but a few of the more common observations seen in cardiac crises.

Examination of the Pulse

Examination of the radial or temporal pulse is the next step in diagnosis; rate, rhythm, and quality are the functional triad of hemodynamic integrity and competence. Here, the very slow high tension pulse of complete heart block is in dramatic contrast with the rapid irregular rhythm of paroxysmal atrial fibrillation with its feeble and oftentimes impalpable pulse. The irregularity of premature beats must be differentiated from certain forms of incomplete block and slow atrial flutter and/or fibrillation; the less rapid but usually regular rhythm in various forms of paroxysmal tachycardia may be confused with sinus tachycardia of thyrotoxic origin. The identification of these changes may frequently be sufficient to make a *presumptive diagnosis of the condition. The discovery of* normal sinus rhythm within the normal rate range is, however, no evidence that cardiac damage is not present; massive myocardial infarction may exhibit no change in rate or rhythm in the initial stage of the syndrome.

Auscultation of the Heart

Auscultation of the heart sounds presents a unique opportunity in clinical interpretation and the physician *should welcome the challenge offered in the* correlation of what may be heard and other physical findings in any given patient. The auscultatory data of the heart is based solely upon what may be heard and identified; the impli-

cation, here, is that the practitioner should use any method which will make these sounds and murmurs, if present, as clear and distinct as possible. The question of a suitable stethoscope is important and brings up a problem which, strange to say, is rarely discussed, but which concerns the use and care of the instrument.

Which is the best type of stethoscope? Since the sole purpose of the instrument is to render the heart sounds and murmurs as distinctly and clearly as possible, any stethoscope which fulfills this specification may be employed. It should fit the ears comfortably and should be free from loose or moving parts which may generate distracting noises; this is particularly true of certain gadgets which are applied to stethoscopes to make them multipurposeful or to extend their field of clinical usefulness. In this connection are the various timing devices attached to the chest piece and the revolving types of diaphragms. Of the variety of chest pieces which have been devised, the practitioner should select the one which most closely approximates his concept of a perfect model, concerning which there are probably as many opinions as the men using them. The type of diaphragm is also a matter of personal preference, scientifically, there is no or little appreciable difference between the older conventional hard rubber and the newer types of plastic discs. Some clinicians use an open or diaphragmless chest piece in order to eliminate skin noises. The open chest piece is a modification of the original ear to chest procedure in examining the heart. The method still has a field of usefulness under certain conditions, the ear to the chest examination may oc-

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In the larger non-portable heart sound equipment, employed in research laboratories, this loss is avoided by complicated electronic equipment; high fidelity stethoscopes are available for tape and wire recording which have a wide range in reproduction of sounds and murmurs.

Auscultation of the heart sounds requires a long period of study and experience if the examiner wishes to secure the maximum clinical information available.

The Time Factor in Auscultation

The *time factor* in auscultation of the heart not only refers to rate and rhythm but also to the length of time required to study the sounds. The 15 seconds, ordinarily employed to estimate the rate, is rarely sufficient to evaluate the auscultatory phenomena; even under optimum conditions a much longer period is necessary. Most cardiologists recommend at least 2 or 3 minutes. Here, patience may be rewarding; changes in the various qualities of the sounds and murmurs as well as an infrequent irregularity may be identified. In this connection, a well practiced scheme or routine of examination will be of inestimable value to the listener. Over the years a number of heart sound codes have been described, all of which have a chief purpose of compelling close attention to details which might be overlooked or missed in a more hasty appraisal; the practitioner should be familiar with at least one. The automatic conditioning provided by such a code will be valuable in every cardiac emergency.

casionally reveal slight heart sound changes or the presence of doubtful murmurs which may be missed by the stethoscope. In men, the method usually can be applied without difficulty; its employment in women has been an age old question with many tangents. The ear to chest method of auscultation may be revealing in certain types of *high pitched, presystolic murmurs in mitral stenosis;* splitting or reduplication of the second sound may sometimes be more easily identified than with the stethoscope.

Electronic Stethoscopes

Diminishing the size of the electronic elements, which have made possible the miniature hearing aid devices, has also produced a trend toward the use of transistor stethoscopes; some of these are only slightly larger than the conventional wide diaphragm chest pieces. Insofar as simple amplification of the heart sounds are concerned, most of these electronic stethoscopes are satisfactory in that they render feeble and distant sounds more intense with a decibel clearance which may be in the range of 4 to 6 times the loudness of the same sounds heard through the ordinary stethoscope. *However, in obtaining such simple amplification considerable fidelity loss occurs since the frequency range of the sounds and murmurs does not follow an equivalent reproductive curve. In other words, increasing the loudness of the sounds is made at the sacrifice of certain harmonic qualities which are characteristic of a given heart sound or murmur;* for example, the crescendo quality of the tight mitral stenosis syndrome may be lost or changed in the miniature amplifying circuits.

Murmurs have long been graded in terms of intensity; Levine was perhaps the first to suggest such a numerical coding of systolic murmurs. Reisman extended the grading to include all murmurs and Opitz later combined the intensity grading with the time periods. Thus, a grade 3 systolic murmur which started early in systole and extended through the mid part could be written as:

First Sound 330 Second Sound 000 First Sound

This might be abbreviated to read:

S_1 330—S_2 000

If the first sound in this patient was graded as 2 and the second as 4, the formula would be:

2S_1 330—4S_2 000

In actual practice the sub numbers 1 and 2 can be eliminated; thus in a given case where the second sound predominated and where there was a grade 3 systolic murmur which ended before the second sound occurred, the simple formula would be:

2S 330—4S 000

All types of heart sounds and murmurs can thus be coded into a simple heart sound formula which has no language barrier. A few common heart sound patterns are given below:

Aortic valvular insufficiency with a grade 3 murmur running throughout the diastolic period and with the first sound louder than the second:

Codification of Heart Sounds and Murmurs

The heart sounds and murmurs lend themselves to a musical scoring system; Rehfisch in 1908 was the first to develop an integrated harmonic scheme of transcribing the heart sounds. He is said to have orginated the concept of heart *tones;* the phraseology of heart tones is widely employed in the European medical schools while in this country and England, the word *sounds* is more common.

Heart sounds and murmurs also lend themselves to a numerical coding system; here, the method is based upon the tonal intensity or loudness of the sounds and murmurs. Since no sense of musical training or appreciation is involved, the numerical system can be used by anyone who can distinguish differences in tonal loudness. The Alsahy numerical scoring method is perhaps the easiest to understand and to employ clinically. Briefly stated, the method requires identification of the two heart sounds at the point of maximum intensity (PMI); an empirical value from 1 to 5 is given to the sounds. Grade 1 is a sound which is very difficult to hear, while grade 5 is a sound which is so loud that it may sometimes be heard at a distance from the stethoscope. Since the first sound indicates the end of the diastolic interval of the cardiac cycle and the second sound the end of the systolic interval and the beginning of the next diastolic, the two phases of cardiac activity can be determined clinically without difficulty. Furthermore, both systolic and diastolic intervals can be divided into 3 equal parts which are known as early, mid, and late systolic and diastolic periods. Murmurs, if present, are indicated in one or more of these 6 time periods.

tant and indistinct murmur to another. There may be an important clinical implication in the two concepts. While the ability to select the correct word to describe a given condition will vary from one individual to another depending upon a host of educational factors, literary expression, and experience, nearly every physician is able to evaluate sounds in terms of loudness or intensity. Thus, heart sounds and murmurs written in a numerical coding system will carry the same information and thought to each reader of the record. As a shorthand method of condensing into a single formula of 10 or 12 digits, the Alsahy method has been widely accepted.

A secondary value of any coding system is in its educational and training potentialities; in order to complete the coding formula, the examiner is forced to concentrate upon the essential data required in auscultation. More time and attention is demanded than for a superficial examination and insofar as these two factors are frequently neglected in cardiac appraisal, the method will serve to make the diagnosis of a given condition more accurate. Physicians trained in the determination of the heart sound formula, automatically think in terms of numerical evaluation on listening to every heart; as an aid to diagnosis the method is recommended to the general practitioner. The criticism that such interpretation of the auscultatory phenomena of the heart loses some of the fundamental characteristics of the sounds is possibly true and it is here, that the musical coding systems offer a more complete analysis of the relative harmonic values.

Only the MPI heart sound coding system has been presented in this chapter: the Alsahy method also includes

3S 000—2S 333

The crescendo murmur of mitral stenosis with loud snapping first sound:

5S 000—2S 123

The Alsahy numercial coding system also provides for splitting or reduplication of the heart sounds; thus the so-called third heart sound which is due to a split second may be written:

2S 000—3Ss 000

Here the small *s* indicates that the intensity or loudness of this split part of the second sound is less than the first part. If the 2 parts are equal in loudness:

2S 000—3SS 000

The Alsahy method was developed as a wartime method for several important purposes; the first was concerned with the attempt to standardize and simplify the written description of a cardiac examination for military medical records. The difficult choice of words, sometimes necessary to present the data about sounds and murmurs, has long been recognized by every clinician and medical teacher; soft, distant, indistinct, blowing, whispering, purring, rumbling, grating, squeaking, musical, machinery, harsh, sharp and brilliant are but a few of the more common adjectives used in medical records. From the point of view of scientific accuracy, criticism is here based not so much upon the picturesque choice of words but as to their exact meaning to others; for example, a soft blowing murmur to one observer may be considered as a dis-

Spirited partisan debate still rages between those who favor the mercury manometer type over the aneroid gauge models. The mercury manometer apparatus, because of its gravity-pressure concept, requires a larger and more unwieldly instrument housing; it is more awkward to carry in an emergency bag. It may also be difficult to manipulate under certain emergency conditions, especially if the patient is lying in an unusual or ungainly position. The relatively large case, when opened, has a tendency to fall over unless firmly placed or anchored on a level plane, this may mean that someone must hold it in a vertical position while the instrument is being used and it is then no longer a one-man apparatus. Technically, this is a violation of Cramp's 4th Rule on emergencies, i.e., the employment of procedures which require the services of an untrained assistant but which are more expeditiously managed by the physician himself. Against these alleged disadvantages are certain important and undisputed facts about mercury manometers; first, they never get out of order unless the mercury is lost. The gravitational factor is not affected by time or use. Secondly, all mercury manometers register pressures up to 300, some as high as 350, many of the older aneroid models only register to 250 mm. Hg. This may be of some importance in certain hypertensive crises.

Blood Pressure Tourniquets

In addition to the determination of blood pressure *per se*, the apparatus has another important function in certain cardiac emergencies; its use as a tourniquet has not been sufficiently exploited. In performing a venesection,

the so-called triangular formula which is concerned with the sounds at the base of the heart at the aortic and pulmonic valvular areas in relation to the apex as well as with the rate, rhythm, and blood pressure, all of which can be expressed in a 2 line basic formula.

To repeat: One of the important time factors in the examination of the heart under emergency as in all other conditions, is the time spent in auscultation. Since treatment is predicated upon diagnosis, a successful outcome may be directly proportional to the accuracy of the information obtained from examination procedures; in acute cardiac crises this may be life saving.

Blood Pressure

In the cardiac emergencies, the determination of blood pressure is frequently an important part of the initial examination; a blood pressure apparatus is one of the essential working tools of the practitioner. Diagnosis in some instances may depend entirely upon the estimation of systolic and diastolic levels; in cardiogenic shock the determination of pulse pressure may have significant implications. In the superhypertensive syndromes, like the pre-apoplectic state with encephalopathy, the effectiveness of immediate emergency treatment can only be indicated by determination of blood pressure levels. Since the apparatus itself has such a vital role in diagnosis, a few observations may be made about the instrument; the practitioner is familiar with certain facts about the apparatus in current use and he may be interested in some considerations experienced over many years of its employment and under various circumstances.

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for conditions later to be discussed, the compression cuff is far superior to the usual rubber tubing or webbed strap for this purpose. The basic concept of Hodges, underlying the employment of a tourniquet for venesection, is the attempt to raise venous pressure by restricting the back flow of the blood stream above the point of vessel puncture. The physiologic optimum degree of compression is a point between systolic and diastolic arterial pressure; pressure above the systolic level will prevent arterial flow into the limb, thereby causing a drop in venous pressure to zero. If, for example, the conventional rubber tourniquet is applied too tightly there will be less rather than more venous flow from the punctured vessel. It may thus require a fine sense of adjustment in order to get the maximum benefits from such a procedure and in experienced hands there may be no great problem. The likelihood of compressing the limb too tightly stems from the more general use of the tourniquet to stop traumatic hemorrhage; here the compression factor must be greater than the systolic pressure. In contrast, the arm cuff of the blood pressure apparatus is a tourniquet with simple and extremely sensitive pressure adjustment. When increased venous pressure in a limb is desired, the pressure in the cuff is set about mid-point in pulse pressure; if complete arterial obstruction or hemostasis is required, the cuff pressure is raised to about 10 mm. Hg. above the systolic level.

Another advantage of the arm cuff as a tourniquet is in relation to post-compression nerve injuries; when a tubing tourniquet is maintained in position for any great length of time there is always the danger of secondary

nerve changes. The arm cuff, because of its greater width, spreads the compression effects over a much larger area; it has been estimated that the ratio here is in the range of 12:1. This does not necessarily mean that the tubing tourniquet is 12 times more likely to produce nerve injuries than the arm cuff but experience has shown that far more such post-tourniquet injuries occur after the employment of rubber tubing than after the arm cuff.

Parenteral Injection Methods

On the spot emergency medical treatment frequently involves hypodermic or intravenous injection therapy. An axiom of all disaster units is that if a given patient requires such treatment, no time can be wasted in preparing the medication. The customary ritual of boiling the syringe and needles in the patient's kitchen has no place, of course, if the emergency locale is on a highway or public building. Here all such parenteral treatment must have been adequately prepared before leaving for the emergency and must be ready for instant use. Most practitioners carry with them pre-sterilized syringes and needles; there are a variety of devices which have been developed by enterprising manufacturers to handle the situation and, the physician, after some first hand experience, will select the type best suited for his particular task.

There is no intent to ridicule the traditional sterilization procedure in the patient's kitchen; when time is not an essential factor and where watching the pot boil does not add to the complexity of the case, the method has undisputed advantages. It is also necessary whenever infusions

or transfusions are indicated; unless the practitioner has prepared infusion apparatus with him, the required tubing and other equipment may have to be sterilized and the boiling pot can not lightly be disregarded for this purpose in lieu of other available methods.

The question of adequately sterilized injection equipment has lost some of its mandatory aspects since the advent of the antibiotic era. In the presence of an acute condition, requiring instant drug injection, no time should be lost in routine sterilization; in cardiac collapse from a number of causes to be considered later, parenteral or cardiac injection can be performed with equipment which may be more or less bacterially *dirty* on the Blake concept that it is better to have an infected live patient than a dead clean one! Antibiotics have rendered the possibility of such infections assuming serious importance almost to a negligible consideration. Sterilization in emergency medicine and surgery has thus lost much of its former significance but, wherever possible, the practitioner should attempt to maintain a scrupulous aseptic technique. The addition of a secondary infection, even though controlled by antibiotic measures, is never beneficial to a patient with critical heart disease.

Cardiac Resuscitation

The amazing progress made in cardiac surgery in the past decade has widened the scope of procedures which the practitioner should and must employ under special circumstances. Chief of these is open massage of the heart in cardiac arrest from various causes. Previously

considered a formidable surgical undertaking, opening the pericardium from below the diaphragm should carry no more hazards than the routine tracheotomy which was performed by every physician at the turn of the century for epidemic obstructive laryngeal diphtheria. Many of these were successfully performed with a doctor's pocket-knife and it is interesting to note that the current medical literature has begun to report similar recoveries from pocket knife surgery in patients with cardiac standstill. While such instances have a certain dramatic public appeal, there are other instruments which should be used whenever life saving operation is indicated. These surgical instruments must now be considered as much a part of the practitioner's armamentarium as his syringes and needles. A few words should be said about these instruments here. Cardiac surgeons claim that a minimum of 4 is desirable, these in the order of importance are: scalpel, long straight scissors, curved clamp, and long straight clamp. Experience has shown that scalpel and scissors are the most essential. It may be pointed out briefly at this time that in cardiac standstill there may be little or no hemorrhage of incised tissues; in ventricular arrest, no blood leaves the heart. Hemorrhage from cut arteries is predicated upon vascular pressure secondary to a *gradient* of ventricular pressure greater than the diastolic closing vector of the aortic valves. When the heart is *in a stand-still* phase, systolic pressure is immediately lost and diastolic pressure is quickly spent. The physiology of cardiac arrest is considered in detail later; it is mentioned here in relation to the necessary equipment to be carried in the emergency bag.

impedance hearing aid wire cords, occupy little space in the assembly. The records produced are legible and, although subject to standardization errors, may be used for emergency diagnostic purposes. Such substandard instrument performance does not vitiate their place in the differentiation of the rapid irregularities; paroxysmal atrial fibrillation with a rate of 220, for example, presents far less clinical hazard than paroxysmal ventricular tachycardia with the same rate. To the practitioner this is an important difference in two apparently similar clinical conditions. In acute coronary occlusion, the need for emergency electrocardiography is less apparent; it may require several hours after the episode before significant changes may be noted in the tracings.

Transfer of the Patient

Finally, some pertinent observations on Cramp's 5th Rule. transfer the patient as soon as possible from the scene of the emergency. Where the episode has occurred in the patient's home, the question of hospitalization for further observation and treatment may involve a number of personal and family problems but, in all other situations, removal to a hospital is a prudent procedure to follow. Most cardiac emergencies, even if successfully treated to the point where the patient has recovered to what might be considered *status quo ante* and is apparently able to continue with his normal activities, may have a tendency to recur in one form or another. The basic pathology responsible for the emergency situation is not likely to have been improved by the acute episode; on

the contrary, it is probable that the cardiovascular disability has been increased even though the individual may deny any such aggravation in the attempt to escape from the implications of having been the subject of emergency attention.

Many cardiac emergencies which appear to be minor disturbances of rhythm or functional capacity may subsequently develop into more serious syndromes; a paroxysmal atrial fibrillation, for example, is sometimes ushered in with several brief episodes which last only a few moments. Cerebral vascular thrombosis may start with a simple attack of vertigo or syncope which may entirely clear up only to be followed some minutes or hours later by massive involvement. An attack of severe angina pectoris may be promptly responsive to emergency treatment with nitroglycerin, permitting the patient to return to his usual activities, but within a short time signs of coronary occlusion may appear.

Medical Records

Practical cardiology, because of the increasing scope of medicine in general, thus has implications not usually considered to be an integral part of the diagnosis and treatment of heart disease. As indicated previously, the astounding development of compensation, industrial and insurance medical care plans require a re-evaluation of the practitioner's role in the handling of any case; the expanded responsibilities must be recognized and must be given the attention demanded under a given set of circumstances. Many doctors, particularly of the older gener-

ation, continue to chafe at the continuing encroachment of these extra-clinical activities believing, as they do, that the time spent in the actual medical care of the patient constitutes the end-all of their professional interest and activity. Under the philosophy of socialized medicine, "medical care" is defined as including all those acts to which the patient is entitled both by tradition and by law. The traditional rights have been well established by many centuries of medical practice; those rights established by law have equal validity and are subject to continual change. To be complete, any presentation of practical cardiology must, therefore, always keep these extra-clinical aspects clearly in mind for all indications point toward more rather than less time consuming so-called "paper work."

A Final Word

A final word about the management of the cardiac emergencies may be permitted here. It is an unfortunately true and regrettably recognized observation that academic knowledge alone is not sufficient to promise a successful outcome in any given acute cardiovascular episode; the additional factors of inborn quickwittedness, ability to make prompt decisions, and above all, the God-given faculty of good judgment under stress, may be the difference between life and death in many conditions.

The following chapters are concerned, first, with some observations about the physical examination of the patient with cardiovascular disease. Then, the various disabilities of the heart and blood vessel system are considered in

their order of statistical frequency as seen in general practice.

In selecting this order of presentation, priority has been given to those subjects which are most likely to command the attention of the practitioner in his every day work. Clinical experience has shown that the rare and unusual conditions which ordinarily occupy large sections of most textbooks on cardiology are consulted not too frequently. When such syndromes are discovered, the problem requires technical studies which are usually beyond the scope of the doctor's office; special hospital facilities and sometimes research institute investigation are necessary to establish a diagnosis and to provide treatment. No apology is therefore offered in this practical application in the consideration of first things first and, it should be repeated again, that this book is written for the medical man and woman engaged in daily skirmish with the formidable forces of cardiac disability both acute and chronic. Here the rewards are great; the personal satisfaction of a job well done, a patient relieved from a critical condition, and perhaps, a life saved, all go to make the practitioner's art and science one of the truly great achievements of this modern age.

CHAPTER II

MEDICAL HISTORY IN HEART DISEASE

"The difference between Art and Science is difficult to define," Dr. David Reisman, the noted Philadelphia clinician once remarked, "but I would say that a good artist knows when to stop whereas a good scientist must never cease in his work."

It is literally true that the examination of the patient with cardiovascular disease never ceases; even the intervals between visits to the clinic or doctor's office must be regarded as periods of observation which are frequently productive of significant information. No other physiologic system of the human organism presents such a wide range of possible clinical and technical investigation. The gamut extends from the simple determination of pulse rate, rhythm and quality, blood pressure, rate and type of respiration, evidence of congestive failure, and color of skin and mucous membranes to the specific examination of all of the various components which make up the heart and blood vessel system. It is not impossible for an experienced clinician to make a diagnosis with no other instrument than a stethoscope and it may still be difficult to arrive at a definitive diagnosis in certain cases even after the most thorough investigation uti-

lizing all of the methods provided by modern technologic advancement.

Every cardiovascular examination thus presents an exciting challenge in which skillful observation, diagnostic ability, and good clinical judgment blended with previous clinical experience, all play an equally significant role. *Moreover, every disturbance of the heart and blood vessel* system is a multifaceted syndrome which is the end result of many known and unknown factors. The identification of the unknown factors is the goal toward which all diagnostic methods are directed but, fortunately, it is not necessary to accumulate all of these in order to start treatment in most patients. The practitioner usually has a sufficiently wide choice of data and methods of obtaining the clinical information required in making a working diagnosis in nearly every case and urgent therapeutic measures can and should be started promptly. As indicated previously, the alarming symptoms of the heart disease like pain, orthopnea with and without pulmonary edema, and palpitation due to the paroxysmal arrhythmias, have a tendency to produce a vicious cycle which may enhance all of the patient's complaints. In these instances, treatment of the symptoms is mandatory even though the specific mechanism responsible for the condi*tion is not well understood or even unknown. Clinical* medicine accepts a delay in instituting treatment under certain well defined instances, such as, for example, in postponing the use of the antibiotics, except in subacute bacterial endocarditis, to identify the predominating organism in a blood culture or withholding the anti-malarial drugs in a suspected case. But, in heart disease there is

no justification in such a delay for diagnostic purposes; in this the author differs strongly with those who would withhold nitroglycerin, for example, in order to determine the pain pattern and time factors of the syndrome. There has recently developed, in high academic places, a tendency to condemn symptomatic therapy on the concept that such procedures are unscientific and add nothing to the overall understanding and management of a given disease process. Additional hazards and positive harm to the patient could result from such "scientific" delay tactics.

The Medical History

The examination of the cardiovascular patient properly starts with the history; the extent and scope of the history is based upon a single purpose, namely, to secure sufficient information to establish a working diagnosis. The history may be extremely brief in certain specific conditions and it may be unending in borderline cardiac disabilities; the history may be the only positive finding in the entire examination, as in angina pectoris, or it may add confusion to an already complicated clinical syndrome. The historical survey may be confined to the patient's previous medical problems or it may be necessary to include his psychosomatic background, marital and sexual problems, social and economic status, as well as remote factors which may have a bearing upon his physical and mental burdens. Heredity plays a not inconsiderable role in cardiovascular disease and time spent in genealogic investigation may yield useful data. It should be empha-

lizing all of the methods provided by modern technologic advancement.

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barrassing moments in every practitioner's busy life are the questions that he forgot to ask at a given difficult situation. Some of these forgotten questions could have subsequent significant implications.

Medico-Legal Aspects of History Records

It would seem to be unnecessary to discuss the importance of making notes in regard to any emergency cardiac case whether treated at home or under extra-domiciliary conditions; since an increasing number of such patients subsequently become medico-legal problems, the practitioner can no longer trust his memory to supply pertinent and specific data required for insurance, compensation, and other mandatory forms several weeks or months after the occurrence. The courts have repeatedly stated that it is the duty of the attending physician to keep and supply such information; the time honored concept that the doctor was too busy treating the patient to be bothered with such nonsense is no longer acceptable; insofar as the patient's rights under due process of law may be jeopardized by failure to keep and supply such information, the practitioner may find himself in an unfortunate predicament unless he can produce the evidence requested. The data should be set down in writing as soon as possible after the emergency situation permits; in court cases, the practitioner as a witness, may be required to state for the record when he made the notations from which he is testifying. It is obvious that the legal importance of such medical records is considerably lessened if they were made some time after the actual occurrence of the emergency.

sized, here, that history taking may be time consuming; not infrequently the time required to obtain an adequate history may be greater than that required for the actual physical examination of the patient. It is not always necessary to complete the history at the first meeting with the individual; it may well be that given time to think things over, the patient will return with pertinent information which could not be remembered at a previous visit. Many patients are ill at ease in the doctor's office or in a clinic; at home with an anxious family surrounding the bedside, the cardiac sufferer may be unable or unwilling to answer certain pointed questions. At such times, the experienced practitioner will sense the unfavorable environment and will postpone further interrogation until a more fortitudinous opportunity occurs. While such withheld information may have significant clinical implications, the physician will usually have sufficient data to initiate the necessary medical care without delay.

Since history taking may develop into a long tedious procedure, it is wise for the practitioner to adopt a simple but all embracing questioning formula. Many such history forms are available; one prepared by the American Heart Association for clinic use is well thought out and rather complete. A number of authors have presented useful forms to follow but the examiner may find it expedient to develop his own routine to meet with the special conditions under which he practices. A printed form is desirable in the office but it may be difficult to use at the bedside; the form is a constant reminder of the leading questions to be asked. It should be memorized by the clinician for use under emergency conditions; not the least of the em-

posed, an employment record showing frequent absenteeism for one or another given reason may be compared with some deductive significance with the record of another patient who has had an uninterrupted working history. It should be recalled here that a medical history is only as valuable as it is factual.

Some Ethical Considerations and the "Third Party"

The subject of questioning is one which demands a forthright answer; the older physician traditionally objected to any question which deliberately forced the patient to make an unintentional exposure or admission of facts and circumstances. The ethics involved were simple; the patient had certain inalienable rights which the doctor must respect. Questioning should go no further than the limits set by the patient himself.

Many changes have occurred in the basic concept of medicine as practiced 75 years ago; many of these changes are related to an expanding social philosophy which considers medical care a communal rather than a private or personal matter. With loss of the previous doctor-patient relationship, a new factor steps into the medical scene; this factor is and will become more and more important. It is known as the Third Party. Who is the Third Party? Jacobson described the Third Party as the corporate power, influence, and accepted legal action of insurance carriers, compensation funds, pension plans, and private and governmental agencies concerned with medical care. In accepting the benefits of this social philosophy, both

One such experience is always a promise to keep better records and the doctor may well profit from the situation which he is not likely to forget.

The Cardiovascular Examination Form

In 1941 when the author was Cardiologist at the Brooklyn Naval Hospital and Director of the Cardiac Consultation Clinic of the Third Naval District in New York, a committee was selected to develop a comprehensive cardiovascular examination chart. This Alsahy C-V Chart consists of a 4-page folder made of heavy weight card stock or bristol board; it is filled out as completely as possible on the first visit. Subsequent examination and progress notes are written on a follow-up sheet which is fastened inside the original folder. Over the course of time a complete record of the patient is available in semi-book form. A copy of the chart form is herewith reproduced; a page by page presentation will permit discussion of a number of important considerations in both the history and physical examination of the cardiac patient.

Significance of Employment History

Page 1 of the case sheet is self explanatory; the item "Employment History" may require a few words. A history of previous cardiac disability may have clinical significance in many instances, particularly in a middle aged man who has denied all symptoms of chronic heart disease; if the preliminary information indicates that he must have had some degree of restrictive activity, perhaps self im-

PRESENT HISTORY

PAIN: Precordial Substernal Radiation
At Rest Walking Stairs Other Max. Dur.

DYSPNOEA: At Rest Walking Stairs Other

PALPITATION: At Rest Walking Stairs Other

CIRCULATORY: Arms Right Left
Legs Right Left

Cerebral Vascular

SLEEP: Hr. Dreams

ONCTURIA:

BOWELS: Gas Hb. Other G-I

APPETITE: C T S A Other

CTA

CLIMACTERIC

LIFE INSURANCE HISTORY

PREVIOUS EXAMINATIONS: Urine Blood
ECG X-Ray BP
Other
ADDITIONAL REMARKS:

FAMILY HISTORY: F M S B D

GENEOLOGY:

OFFICE RECORD	FILE CODE	IN	OUT	RECORD COMPLETE

FORM NO. 40 - [illegible]

ALBERT SALISBURY HYMAN, M.D., F.A.C.P.

NEW YORK

Cardiovascular Examination No.

Name ______ Sex ______ Age ______ S M D S

Residence ______ Phone No. ______

Business ______ Phone No. ______

Referred by ______ Report to ______

Chief Complaints ______

Past History ______

Childhood Conditions ______

Rheumatic ______

Luetic ______

Recent Illness ______

Surgery ______

Accidents ______

Employment History ______

X-RAY MENSURATION OF HEART AND AORTA

Width of Thorax	Pelvis	C P Index	
Width of Aorta	Predicted	Diff	%
Transverse Diameter	Predicted	Diff	%
Surface Area	Predicted	Diff	%
Cardiothoracic Ratio	Predicted	Diff	%
Angle Long Axis	Correlation		
Correction for Over Weight Factor			

EYE GROUNDS: Right Left

Peripheral Vascular:

SENSITIVITY TESTS: Lewis Libman Grater

Xyphoid Up Down Other

BREATH HOLDING TESTS:

EXERCISE TOLERANCE TEST: Wt Stairs Climbed Work

	BEFORE	AFTER	2 MINUTES	5 MINUTES	OTHER
Pulse					
Resp					
BP					
Pain					
Dyspnoea					
Palp.					
Vertigo					
Irreg.					
Murmurs					

ELECTROCARDIOGRAMS: Conventional

Unipolar

HEART SOUND EXAMINATION:

LABORATORY: Urine

Blood

Other

OTHER TESTS:

DIAGNOSES: 1

2

3

TREATMENT:

ORIGINAL WORK SHEET FOR CARDIOVASCULAR EXAMINATION

WT Last Year Most @ Normal Diff %

HT: Last Year Age Apparent Diff

COLOR: Under A U Skin *MM* *Other*

DYSPNOEA: Rest Simple Test Max Test

INSPECTION: Pulsations Thrills Other

HEART SOUNDS	QUALITY	INTENSITY	OTHER
Apical 1st			
Apical 2nd			
Pulmonic 2nd			
Aortic 2nd			

RHYTHM: Vagal Effects

Irregularities

RATE: Apex. Radial Recumbent After Exercise

MURMURS:

LUNGS:

ABDOMEN: Liver Spleen Fluid

LEGS

BLOOD PRESSURE: Standing Rt Arm Lt Arm Handedness

Recumbent Quick Standing Diff

Rt Leg Left Leg Other

OSCILLOMETRIC READINGS: Lt Arm High Low Rt Arm High Low

Lt Leg High Low Rt Leg High Low Other

VITAL CAPACITY: Up Down Diff %

Reduction based on Wt on Ht Average

FLUOROSCOPY: REMARKS

Dyspnea

Dyspnea is a common symptom in heart disease; in contrast to pain, many patients have a tendency to sublime the subjective reaction of breathlessness by rationalization. The fact that dyspnea occurs on climbing stairs is accepted as the natural effect of growing older or heavier; the belief that "everybody gets out of breath climbing stairs at my age," frequently conceals a marked grade of exertional dyspnea. The examiner should be very specific in his questioning about the onset of this symptom; patients enjoy a play on words in this connection. An individual may deny "breathlessness," but will admit readily to being "short of wind" or he may deny "short of wind" but confess to "heavy breathing." The chart provides for notes concerning dyspnea at rest, after walking (how far?), on stair climbing (how many steps?). Dyspnea after running, playing golf or other sports, swimming, shower baths, dancing, coitus, and post-prandial should be noted under item "other."

Palpitation and Heart Consciousness

Palpitation may be difficult to define for the patient; subjective sensations which are referred to the precordial area or even to the entire left chest may be interpreted by the individual as arising from the heart. Strictly defined, palpitation is the sensation of heart consciousness or the awareness of the beating heart; it may occur normally at

the patient as well as the doctor have *given up what is* now considered "the fetish of inalienable rights." What has all this discussion to do with history taking in a patient with heart disease? The courts have ruled that no ethics are involved in getting at the truth and any question may be asked provided the intent is to secure such information.

Pain

We turn now to Page 3 of the chart form; this is concerned with a detailed history of the present status of the patient. The first item is in regard to pain, its distribution pattern and the common factors which are likely to cause the pain to develop. The difference produced by various types of physical activity may be significant; the painful symptom may vary at rest, on walking, and after climbing stairs. The extent of the walking compared to the number of steps climbed to produce the same pain is of interest. Pain produced by psychogenic factors should be noted in detail; painful reaction to anger or fear may be more intense and longer lasting than after considerable physical effort. An attempt should be made to induce the patient to assume an objective evaluation of the pain; item "Max. Dol." or maximum dolor estimation has already been presented. The change in attitude from subjective to objective evaluation may yield unexpected and surprising results.

after the attack has subsided. True Raynauds's disease with painful snow-blanched fingers has not been a common finding in our experience; it has been discovered in the adolescent group and it is more frequent in girls than in boys. A recent study indicates that the syndrome is definitely less frequent today than 50 years ago and more common in the underprivileged than the better housed and nourished groups.

Circulatory disturbance of the legs are far more common than in the arms; intermittent claudication by day, and "night cramps" while in bed, should be carefully discussed with the patient. While there is some difference of opinion concerning the overall significance of this symptomatic finding, it should be regarded as a vascular equivalent of angina pectoris. The association of the two syndromes is common and the treatment of both is, in many cases similar. Insofar as peripheral vascular and coronary artery disease are manifestations of the same basic pathology, the development of one may presage the eventual occurrence of the other. Disturbances of the venous circulation of the legs should be noted in the history.

Vascular Headaches

Cerebral vascular symptoms include headaches, vertigo, syncope, and episodes of unconsciousness. In general, headaches are more common in hypertension and dizziness in hypotension. Localization of the headache may have clinical significance; posterior and occipital headaches are more likely to have a vascular background than frontal or temporal. Note should be made concerning the

night while lying on the left side and it is frequently noted in normal persons after exertion. There are many non-cardiac causes for heart consciousness; the chief of these are gastrointestinal. The gas filled stomach or colon may not only produce palpitation but also pain in sympathogenic responsive subjects. Sinus tachycardia leads the list of identified causes, followed by premature beats, atrial fibrillation, atrial flutter, paroxysmal tachycardia, second degree heart block, and a small miscellaneous group of cardiovascular conditions like marked hypertension with sinus bradycardia. In obtaining a history of palpitation, the circumstances of its occurrence are important; items "At rest," "Walking," and "Stairs," indicate the grade of effort to produce certain types of heart consciousness. "Other" is a measure of psychogenic influences, postprandial effects, or other special conditions.

Circulatory Symptoms

Circulatory symptoms in cardiovascular disease may masquerade under a wide variety of subjective complaints; the differential diagnosis from arthritis, neuritis, rheumatic complaints, orthopedic problems like the shoulder-arm syndrome and spinal deformities as well as many other disabilities may be difficult and at times impossible. In the arm, tingling and numbness of the fingers, usually on the left but occasionally on both sides, is not uncommon in peripheral vascular disease; it may occur both with and without anginal syndrome. In some anginal patients, the left arm symptoms may preceed the precordial pain pattern, while in others, it may remain for some minutes

stands in contrast to the nausea and vomiting which may occur in cardiac patients who have been overdigitalized. Vertigo is a common symptom in many diseases; its association with cardiovascular conditions must be clearly defined.

Epistaxis

Epistaxis should be noted in the history; *nosebleeds* may occur both in high as well as very low blood pressure although it is far more common in the hypertensive group. In the latter, it may be considered as having a teleologic significance since the blood loss is in effect a physiologic venesection. Insofar as such blood loss may have a prophylactic aspect in relation to the possible prevention of a cerebral accident, epistaxis may not be an unmixed blessing but frequent and profuse hemorrhage must be kept under laboratory observation. A marked drop in both red cells and hemoglobin may add complications to an already overburdened cardiovascular-renal problem. Unless specifically asked the question, patients do not report epistaxis; the connection between nosebleeds and hypertension may appear to be remote.

Sleeping Habits

Sleeping habits in the cardiac patient have not been sufficiently emphasized by most authors yet, *insomnia* is a very common complaint. There are many reasons why individuals with heart disease cannot sleep; nocturnal dyspnea, palpitation, pain, radiation circulatory syn-

apparent depth of the headache; vascular headaches are usually described as deep within the head compared to the more superficial localization of other types. However, temporal arteritis may have a superficial skin distribution. The frequency and duration of the headaches as well as the time of onset during the day or night, may have subsequent significance. The response to the common headache remedies should be noted.

Vertigo

Vertigo and giddiness may result from postural hypotension; while this symptom may occur in normal individuals with poor physical fitness, it is frequently noted in chronic heart disease. On arising suddenly from a reclining or sitting position, systolic blood pressure may fall as much as 30 to 40 mm. Hg.; the condition is said to be due to a loss of vasomotor control secondary to sclerotic changes in certain arterio-ganglionic reflex centers. On questioning, the patient may state that when he gets out of bed at night, for example to urinate, he becomes unsteady and dizzy when he starts to walk; he may find it necessary to hold fast to a chair or other bedroom furniture for a few moments. In extreme cases, such patients have fallen to the floor and in several instances a presumptive diagnosis of a "mild stroke" has been made. The differential diagnosis may be suggested on repeating the procedure during the physical examination. Vertigo from high blood pressure is more constant and is usually associated with headache. Vascular types of vertigo, regardless of their severity, rarely produce nausea and vomiting; this

For example, a normal individual usually sleeps 8 hours, gets up promptly, and has no reclining rest during the day. His sleeping formula or pattern could be expressed as: 8(8) + 0. This can be compared to a patient who states that he sleeps only 4 hours but remains 10 hours in bed; he also rests about 3 hours during the day. This may be written as 4 (10) + 3. Changes in the sleeping pattern are easily noted and the results of sedation or other medication are accurately followed.

The Fear of Going to Sleep

Insomnia, which is chiefly due to the fear of going to sleep, may also stem from the dream factors responsible for the syndrome of angina decubitus, the patient, while lying motionless in bed, may nevertheless experience a severe episode similar to exertional or effort angina pectoris. Physiologists have long shown that the activities of dream life may be misinterpreted by the autonomic nervous system; it is a normal experience to have dreams so vivid that the individual awakens with tachycardia and breathlessness. Many anginal sufferers who formerly developed severe seizures after exertional effort and who have since learned to avoid such activity, may awaken with pain as the result of a dream sequence in which the previous activity is repeated. This is illustrated in the case of a 62 year old bank executive who lived on Staten Island but who had his place of business in Manhattan; he traveled by ferry and it had been his habit on many occasions to run across the wharf and down the gangplank just as the boat was about to pull out. In recent months he began

dromes, are but a few of the major obstacles to a restful night. Insomnia ranks high in the list of woes related by patients among themselves but it may be omitted when in the doctor's office; real and fancied loss of sleep may, however, present a baffling problem in certain types of heart disease where congestive failure and the possibility of acute pulmonary edema is present. Individuals who have suffered bouts of nocturnal dyspnea with or without pulmonary edema may subconsciously be *afraid* of going to sleep, yet, at the same time, they may complain bitterly of "Lying awake all night." The item "Ht" on the chart is in regard to the number of pillows used or the height necessary to permit comfortable breathing; this may be a revealing question in that it will reveal inconsistencies in the patient's statement that he has breathlessness but he can sleep flat or with only one small pillow. The reverse is also true; the individual denies dyspnea but uses 3 or 4 pillows at night.

The Harvard Sleep Formula

Since the sleeping habits of the cardiac patient may have some bearing upon treatment a more or less accurate estimation of his sleep and resting periods is essential. Answers such as fair, poor, very bad, no sleep at all, have little significance; one individual's "poor" may equal another's "good" and "very bad" could be "fair" to a second patient. The Harvard Sleep Formula is a simple but informative statement; it consists of the hours spent in sleep, the hours spent in bed and the hours utilized for resting during the day.

competition when she was carried off the skating rink and required hospitalization. Repetition of this dramatic incident was constant in the dream sequence and she would awaken with dyspnea and palpitation; on several observations made by her sister who was a nurse, the pulse rate was found to be as high as 160.

The content of dream sequence is not always recognized by the patient, who even may deny dreaming at all; where the physician may have cause to suspect that nocturnal episodes of pain, dyspnea, palpitation, and even acute pulmonary edema may be related to such a stimulus, the time spent in questioning the individual will be rewarding. There is no intent here to suggest that all such attacks are due to an autonomic dream reflex although many psychiatrists believe that this unconscious motivation factor is the trigger mechanism largely responsible for the development of the syndrome. Wolf points out that the chief factors, usually considered to be productive of such nocturnal attacks, like slowing of blood stream velocity, drop in blood pressure and decrease in pulse rate—all of which result in a lessening of blood volume exchange—are a common denominator in any given cardiac patient whenever he is asleep. The added factor required to trigger the mechanism is the stimulus of exertional activity which in the case of a dream content is a response of the autonomic nervous system.

Nocturia

In older patients, nocturia and insomnia may be related; if sleep is broken by a full bladder, the individual may not

to have increasingly severe attacks of anginal pain after this morning performance; he finally experienced a coronary episode and retired from his work. About a year later, he reported that he was awakened at night by extremely severe attacks of angina pectoris which, while relieved by nitroglycerin, were nevertheless causing a deterioration in his cardiac status. The explanation of his nocturnal episodes remained obscure until he admitted, on close questioning, that he was troubled by a dream which had a tendency to be repeated several times a week. In the dream sequence, he was again back in the habit of charging down the gangplank in the dramatic attempt to catch the ferry; he would awaken with the same breathlessness and with the identical pain pattern as that previously experienced in actuality. A change in his sleeping habits eliminated these attacks.

Patients with a low effort tolerance, who experience marked breathlessness, even on slight activity, may have episodes of nocturnal dyspnea which are precipitated by dreams. Here, the dream sequence contains one or more of the forbidden activities; a music teacher, age 44, with mitral stenosis and atrial fibrillation, was plagued with repeated attacks of nocturnal dyspnea, some of which were accompanied by early pulmonary edema. Close questioning disclosed the occurrence of dreams in which skating was a predominating activity; in her younger days she had enjoyed figure skating and racing. She had completed successfully in a number of tournaments but eventually she was forced to give up all these activities because of increasing cardiac disability. She had vivid memories of the marked breathlessness which occurred during the last

If the trip to the bathroom is a long one, the simple expedient of a bedside urinal will be appreciated by patients, both men and women. The question of nocturia as a needless expenditure of energy in individuals with low marginal reserve cannot be overemphasized. Getting in and out of bed several times during the night may spell the difference between a normal and retarded convalescence in certain cases.

The Gastrointestinal Tract

Disturbances of the gastrointestinal tract have a very direct bearing upon the cardiovascular system both in health and disease; acute dysfunction of the stomach is always a constant threat in heart disease. Such disturbances may either result from the primary mechanism of congestive failure or from the secondary effects of medication. Many of the drugs used in the treatment of heart disease are gastric irritants—digitalis, aminophyllin, the iodides, mercurials, nitrates, and some of the anticoagulants—all may produce a number of undesirable by-effects. Nausea, vomiting, anorexia and gas may be serious complications during an acute coronary thrombosis or during a period of congestive failure. Disturbances of the colon and constipation may likewise aggravate a critical cardiovascular problem. Questions concerning the patient's bowel habits, the extent of gastric distress, heart burn, and abdominal pain may be revealing; gall bladder and coronary disease are constant companions in many middle aged persons. Angina pectoris, peptic ulcer, and chronic colitis are also a well known triad; the possibility

recognize the accumulative effect of repeated trips to the bathroom during the night. The causes of nocturia are not well understood. In men, the prostate has been assigned a leading role but women of equal age may also experience the same symptom. A greater volume output of urine at night than by day is common in cardiac patients; this may be due to the postural effects. King has studied the orthostatic factors in urinary output; the venous pressure gradient factors are less in the recumbent position. Where venous pressure is increased, as in both foreward and backward heart failure, a more favorable vascular plane permits a better renal function. The effect of body position may be tested by determining the relationship between diurnal and nocturnal urine output. The normal subject has a 2:1 ratio, that is, about twice as much urine is excreted by day than by night. In older non-cardiac individuals the ratio approaches a 1:1 ratio, while in patients with heart disease the relationship is usually reversed with a 1:2 or even 1:3 ratio. The use of diuretics may complicate the output ratio; nearly all of the oral diuretics require from 10-16 hours to reach full therapeutic response. Unless the drug is given very early in the morning, for example before 7 A.M., there is likelihood that the greater volume output will occur during the night. Insofar as frequent nocturia may be a disturbing factor in certain patients, the regulation of diuresis assumes some clinical importance.

Prostatism is common in the older male patient and simple palliative and expectant measures should be adopted if nocturnal frequency is present. Limitation of fluids after 6 P.M. as a routine procedure may lessen nocturia.

general agreement among cardiologists that angina pectoris, coronary disease, hypertension, and congestive failure are not benefited by smoking. Like all other habits, patients tend to minimize the amount of tobacco consumed; regardless of the compelling arguments in favor of its continued use, the practitioner should be firm in prohibiting further smoking.

Alcohol, on the other hand, has many advocates; Brooks once said that he had never seen an alcoholic with angina pectoris. Alcohol, in excess however, does not spare the vascular tree and the question of "dosage" must always be carefully considered in each patient. Alcohol is contra-indicated in hypertension and in any cardiovascular-renal syndrome. It may have value in some of the peripheral vascular conditions; its chief claim as a therapeutic agent is in relation to its traditional use in the angina pectoris of elderly men. Alcohol has its social and psychogenic aspects and insofar as this may contribute to the comfort and well being of a cardiac patient, the practitioner in his wisdom will set the prescribed limits for its use.

Catamenia

The menstrual history frequently has some importance in those patients who exhibit endocrine disturbances; young women with rheumatic valvular heart disease have a tendency to experience attacks of palpitation and pain during or just before the menstrual cycle. The palpitation or heart consciousness is usually due to a sinus tachycardia with rates running as high as 130; the mechanism is probably a chain reaction involving the pituitary, adrenal,

of hiatus hernia as the cause of cardiac symptoms must be kept in mind. The history of previous and longstanding gastrointestinal problems is not uncommon in cardiac patients and the practitioner must constantly evaluate in any given disability of the heart a probable relationship with some known or unknown gastrointestinal syndrome.

The eating habits of the patients are always important; with diet assuming an increasing scientific status in the treatment of many of the cardiovascular disturbances, questions in regard to salt restriction, fat and cholesterol regulation, balanced caloric intake and fluid limitations may have significant implications. Many individuals on self-imposed or self-modified diets are ruled by facts and fancies which are frequently odd and bizarre; Libby has pointed out that the higher the intellectual level of the patient the more likely he is to fall prey to fantastic dietary beliefs. Clinic patients as a rule adhere strictly to a given diet. Eating habits may thus provide a clue toward solving certain symptoms which the patient may present.

Coffee and tea still remain debatable problems; the recent upswing of "coffee breaks" in the business and industrial world has definitely increased the amount of caffeine consumed; added to this are the caffeine fortified cola drinks which are everywhere available. A recent study indicates that many persons are thus taking from 35 to 50 grains of caffeine a day; in responsive individuals this may produce a variety of cardiovascular symptoms.

Smoking also occupies a stage of excited controversy; while there may be no clear cut relationship between smoking and lung cancer, the clinical evidence against smoking is well established in heart disease. There is a

during this period. Data concerning the climacteric have a definite place in the patient's history. While less well defined, and sometimes difficult to establish, the male climacteric may be the starting point for a number of cardiovascular complaints, heart pain is the most common symptom and it is usually of the effort type. Irritability factors and changes in personality, if recognized, may offer a clue concerning the background for such disturbances. No correlation has been found between the development of coronary heart disease and the endocrine disturbances of the male climacteric although the concept of combined androgen-estrogen therapy is largely based on functional insufficiency of glandular balance. Some authors believe that a man with marked symptoms of the climacteric is a less likely candidate for coronary disease than one who passes through the period with no evident symptoms. In the middle aged man, questions directed towards the climacteric have clinical implications.

Life Insurance History

The general wide acceptance of life insurance has provided an important new source of information in the average patient's history. Since all life insurance requires a *physical* examination, the date of the policy marks a specific milestone in an individual's medical history.

Rejection of the individual's application for life insurance carries equally important implications. A man at the age of 35 was found to have advanced signs of mitral stenosis but he denied knowledge of this syndrome; he admitted, however, that when he was 23 years old he was

and thyroid glands. Premature beats are also common and extrasystolic *arrhythmias occur. So-called idiopathic* paroxysmal superventricular tachycardia and atrial fibrillation have been noted in such rheumatic patients. If the woman presents a history of any of these disturbances, a review of the menstrual history in relationship to the development of these episodes may be revealing.

In older women, usually in the 40's, menstruation may be associated with a considerable rise in systolic blood pressure with its accompanying headaches, dizzy spells, and sometimes precordial pain. Palpitation and irregularities are also not uncommon in the valvular heart disease group. Establishment of the correlation here will be helpful in treatment.

The Climacteric

During no other period of a patient's life, are the symptoms of cardiovascular disability more likely to be enhanced than during the climacteric; this observation applies to both men and women. The menopause in women is, however, more dramatic and, when associated with marked endocrine changes, may cause a wide variety of cardiac symptoms. The chief of these is increase in blood pressure, sometimes to extremely high levels in the range of 250/100, and the increase in the pre-existing complaints. In this group of patients are those who have repeated attacks of paroxysmal tachycardia, atrial flutter, and/or fibrillation, and extrasystolic arrhythmias. The entire syndrome of mitral stenosis may be aggravated during the climacteric and congestive failure may first appear

questions about these various types of physical examination, considerable informative data may be lost.

PREVIOUS LABORATORY EXAMINATIONS

Most physicians inquire about data secured from laboratory studies made prior to the first meeting with the patient; a small group hold to the concept that they would rather not be influenced by such information and would prefer to perform their own tests. It is true, that unless specifically written down, some patients may offer inaccurate data. Where figures are important, as in blood chemistry examinations, basal metabolism tests, and blood counts, it may be unwise to trust to the patient's memory but in general some clue can be obtained from even the most unintelligent person. The result of technical graphic tests, however, like electrocardiograms, ballistocardiograms, phonocardiograms, and arteriograms, must actually be seen by the practitioner; no word of mouth, even from one physician to another, can replace personal study of a given record. This holds true for X-rays of the heart and chest; Williams was fond of quoting that one glance at an X-ray film was worth a million words.

There is a definite place in medical history taking for laboratory reports which are brought in with the patient; such factual information should be faithfully recorded on the chart with the dates when the tests were made. Assuming that the examinations have been performed by competent technicians under approved supervision, there should be no debate about the facts but every physician reserves the right, as well as the duty, to question the in-

rejected for life insurance because of a "heart murmur." If such a murmur was found at 23, it may be assumed that this was the result of adolescent rheumatic infection and that the lesion was probably present for a number of years. The condition was not "new" as he contended and the remainder of his problems could now be appraised in the light of this added information. The onset and possible duration of hypertension can also be estimated from the policy date rejection for high blood pressure. Patients, as a rule, have a tendency to forget life insurance rejections; they frequently state that the valvular condition, hypertension, or irregularities of rhythm, for which they are now seeking medical attention, is of very recent origin. When their life insurance history is brought into the questioning they may admit readily that 5, 10, or even 15 years before they were refused a policy for this very complaint.

In lieu of life insurance, the same data may be secured from records of military service; an important date is fixed, for example, when a candidate for the armed services is rejected for cardiovascular causes. Civil service appointments also require a physical examination in most departments; candidates for police, fire, sanitation, and other positions in public services also must pass an examination. Many large industrial organizations require a pre-employment test of physical fitness. In short, the number of physical examinations for various purposes tends to increase; information about the results of these previous medical appraisals should be obtained in every patient if possible. It is worthwhile to repeat that, while patients usually reveal their previous medical history without too much prompting, unless the physician pointedly asks

environmental factors play is not as important as recognizing that the combination has threatening possibilities.

The family history in every patient with established or suspected coronary, cerebral, or peripheral vascular disease, should be carefully analyzed; hypertension, insofar as renal artery pathology may be responsible in whole or in part for the syndrome, may well be included in this group. Time spent in tracing the medical geneology in as many generations as possible, will give added weight to the practitioner's diagnosis and prognosis in patients who may tend to scoff at the doctor's interest in what his grandfather died from many years ago. An illustrative case is that of a reluctant baseball player, age 38, who was referred by the club physician because of precordial pain, experienced during the training season. Complete cardiovascular study revealed no objective findings of heart disease either at rest or after the exercise tests. The family history, however, disclosed that his father and two uncles had died in middle age of coronary disease, the paternal grandfather and his brother also succumbed to this condition, and patient's own brother was just convalescing from a coronary episode. When these facts were brought to attention, the gravity of the situation needed no further amplification. It would have been poor medical judgment to permit him to continue with the strenuous activities demanded by professional baseball not withstanding the apparently normal cardiac examination; here the history of precordial pain considered within the framework of the significant family history outweighed the apparently normal physical examination.

terpretation. In electrocardiography, all previous tracings are important, regardless of when taken, and previous roentgenograms showing the size and configuration of the heart and aorta are useful for comparative information. It should be a rule that no previous laboratory records should be destroyed.

Family History

There are few medical conditions where the family history is of more significance than in cardiovascular disease; while hereditary factors have not been established with the scientific definitiveness demanded by genetic research, there is much clinical data to indicate a family tendency towards certain types of heart and blood vessel disability. This is especially true in the vascular degenerative pathologies; it is not uncommon to find a number of coronary individuals, for example, in several generations of a given family. Hypertension and cerebral accidents frequently predominate in family groups; peripheral vascular disease, to a lesser extent, may also have a high family incidence. The factors of environment, unquestionably, play a supplementary role in enhancing the likelihood of vascular pathology; the eating, living, and personal habits represent a common denominator in most of these family groups. The concept that high calory, high fat, and high tobacco consumption in an individual with a tendency to atheromatous disease constitutes the profile of a probable candidate for coronary thrombosis has gained wide acceptance in the past decade. The question of how much the inherited tendency and how much the

sound recorders, pulse wave or arteriogram machines, circulation time material, kymographic attachments, catheterization equipment, strain gauges, exercise tolerance equipment for cardiopulmonary function tests and the special electronic equipment required for isotope work. The biochemical aspects of the examination may be included in the fully equipped heart station but in most institutions the general laboratory services do the required chemical procedures.

It should be emphasized again that in actual practice few patients require the entire gamut of technical investigation. Perhaps less than 10% of any large cardiac clinic or private office is concerned with cases so complicated or so obscure that they require such scientific examination and it is doubtful that the practitioner should burden himself with expensive equipment which will be used infrequently. Moreover, if the given case is so complex that it requires these several tests, it is likely that the data obtained will also demand the attention of a group trained in this special field of investigation.

The practitioner should at the outset provide himself with a well thought out work sheet for the cardiovascular examination; many are available. A work sheet has an important function in constantly reminding the physician of the blank spots in any given examination but it is not necessary to complete the tests at one time; it may, in fact, be more informative to collect the data over several visits. This applies especially to the exercise tolerance tests only one of which should be performed on a given day.

CHAPTER III

THE CARDIOVASCULAR PHYSICAL EXAMINATION

It has been pointed out previously that disturbances of the heart and blood vessel system may present a wide variety of multifaceted syndromes. The purpose of the physical *examination is the accumulation of objective* data; the scope and extent of the examination of these syndromes is based upon securing the necessary information required to make a satisfactory diagnosis upon which treatment may be initiated. Since the cardiovascular system readily lends itself to definitive technical investigation, many avenues of approach are possible and in most instances the practitioner will only be limited by the type and potentialities of his equipment. Every physician has the simple basic tools for diagnosis; stethoscope, blood pressure apparatus, a watch, and his own experience, may be sufficient equipment in many cases to make a working diagnosis; few physicians possess the complete array of apparatus which is today available for cardiovascular study. In the order of their relative practical importance, I would say that an electrocardiograph comes first, followed by a fluoroscope or other X-ray equipment, then vital capacity apparatus, oscillometric devices, heart

explanation that the loss is water and is better out of the body than in, will allay their fears that the condition is worsening rather than getting better.

A weight history of the individual should be obtained at the first visit; the weight a year previously, and his greatest weight, and at what age, may be important. For example, if the present weight is 160, a year ago 162, and the greatest weight is 165 about 10 years ago, the clinical implications are far different than a present weight of 165, one year ago 140 and the greatest weight is the present one. Likewise, a present weight of 160, a year ago 140, and 10 years ago 180 has a far different significance. The normal weight obtained from tables can usually be used as the "dry weight" of the patient; the dry weight is the goal toward which the dehydration therapy is directed. Often the calculated normal weight is higher than the actual dry weight; individuals with long standing edema are likely to suffer from undernourishment and a real loss of body weight occurs. Diuresis washes out electrolytes and many other substances like vitamins and certain endocrine complexes. Attempt should be made to correct the difference between the dry weight and calculated normal weight if this is more than 10%.

Height

Data concerning height is far less important than weight; except in growing children and in young adults height determinations are chiefly of interest in relation to the calculation of the various measurements of the body and heart in such procedures as the cardio-body index

Weight

Weight of the cardiac patient carries two important pieces of clinical information; the first, of course, is the determination of his comparative position in the height and age data of the group. In other words, whether he is lighter or heavier than the average individual of his type or body build. Standard age, height, and weight charts for the various body types should be at hand in every practitioner's office. The second importance of weight is its employment as a measure of water retention in all forms of congestive failure. The success or failure of diuretic therapy is readily recognized by gain or loss in weight; subject to an inconstant error, a loss of about 2 pounds suggests a favorable water output over intake of 1000 ml. Patients on a dehydration routine should be weighed every day at the same time and under the same conditions. A comparative test of both measuring intake and output of fluid with the loss of weight is advised in such patients for at least one day; as an object lesson, it increases interest in what the physician is attempting to do and the patient's cooperation is usually improved. Many individuals in the early phase of treatment may be reluctant to measure urine as well as keeping the notes required for fluid intake over a 24 hour period. Sudden or daily weight changes in cardiac patients are more likely to represent changes in water balance than actual body tissue alterations. Great drop of weight during a period of diuresis may cause alarm in some individuals especially if the loss is in the range of 20 to 30 pounds; the

of the skin and mucous membranes may be pathognomonic in certain types of cardiovascular disease. In those lesions associated with poor pulmonary function, cyanosis of various grades occurs. Cyanosis is marked in congenital anomalies involving the pulmonic artery both with and without shunt disturbances. Cyanosis is characteristic in advanced mitral stenosis; here the blueness of the skin softened by the rose blush of the feminine cheek presents the "high color complexion" familiar to the Victorian school of artists. Cyanosis of the lips occurs in cor pulmonale and is not uncommon as one of the early signs of congestive failure.

The skin of the body as well as of the face and neck should be scrutinized; local areas of cyanosis, particularly over the upper chest, may be more evident than on the face. Cyanosis of the hands and feet occurs in peripheral vascular disease but it is also seen in mitral stenosis. Cyanosis may occur in skin areas over massive edema; the legs and lower abdomen frequently show a dusky blueness in patients with anasarca.

Redness and flushing of the face and neck is common in hypertension; the "florid complexion" in hypertensive alcoholics is well known in the cardiac clinic. Such floridness should not be confused with acne rosacea or telangiectasis faciei; White's pressure test is a simple distinguishing measure. The redness and flushing seen in hypertension may disappear under finger-pressure whereas these skin diseases of the face remain red.

Infrared and ordinary red goggles, such as those used in X-ray dark room adaption, are useful in examining the skin and mucous membranes; the blue range of the spec-

surface area of the body, and the cardiopulmonary index tests. The height factor is also used in tests of vital capacity and certain function tests later to be described.

In every patient, however, the present height should be compared with previous determinations, preferably of a year ago. Most patients know their height although there is a general tendency in men to overestimate it. It should not be necessary to state that all height determinations are made in bare feet; the current high heeled shoes in both men and women may give an apparent increase in height as much as 2 to 3 inches.

Height tends to decrease with advancing age; this is due to a number of factors. In patients with heart disease, and particularly in those with associated pulmonary conditions, and compensating thoracic changes, there is a forward bending of the spine with a lessening of the total body height. Over the course of years this loss may be in the range of 1 to 3 inches. Ordinarily, height determinations should be made once a year.

Color

Color of the skin and mucous membranes should be noted in some detail; the observation must be made under lighting conditions approaching normal sunlight or a white light source within the 3000 Angstrom unit range. Fluorescent illumination which is in increasing use in most clinics and offices emphasizes the blue range of the spectrum and the ghastly appearance given to the skin and mucous membranes of the lips, mouth, and tongue permits no estimation pallor or cyanosis. Color changes

such a simple effort test becomes a valuable yard stick in the measurement of respiratory function. Marked differences in resting and after simple effort may be used on subsequent examinations as a comparative index of treatment and prognosis.

Maximum effort tests should not be performed on the first examination; such tests employ 5 to 10,000 foot/pounds of work. In patients with a poor recovery phase the remainder of the examination may yield inaccurate data; an abnormal response to exercise may be reflected in nearly all of the special tests later to be described. Indeed, the entire concept of the function tests is based upon these changes at rest and after effort. The initial cardiovascular work sheet is concerned with the individual at rest; with this as the subject's own standard, the function tests become more informative.

A final word about dyspnea as a symptom; it may be recalled that confusion about the word "breathlessness" is common. It may be wise for the practitioner to discuss its appearance in detail with the patient. The simple test to produce dyspnea may be used; if the subject is showing signs of dyspnea these signs should be pointed out. Certain patients, even when struggling for breath, may deny breathlessness; it is therefore important that patients understand the doctor's interrogational purpose.

Inspection of the Cardiovascular Patient

Inspection of the cardiovascular patient covers a wide field; literally, it means examination of the entire surface of the body. This requires stripping the patient of all

trum is exaggerated when viewed through red lenses. Blue rays appear to be black; small subcutaneous venules can be thrown into relief against the skin illuminated by white light. Lips that appear to be normal in color may show some cyanosis when so viewed. In congestive failure a network of superficial veins over the front of the chest may be clearly visualized. After some experience the practitioner may be rewarded by examining selected patients with such red glasses.

Dyspnea

Dyspnea is both a subjective and objective symptom. There may be a strange lack of correlation between the two. Patients may complain of breathlessness yet the respiratory rate may be slow and within normal limits. The reverse is common; there may be no complaints of dyspnea but the individual is obviously struggling for breath. The respiratory rate is important in most types of heart disease; it is usually increased in cardiac disability. The rate should be obtained without the patient's knowledge; respiratory rates are subject to considerable voluntary control. Many individuals have a tendency to either slow down or speed up respiration if they are aware that the rate is under observation. The basal rate at rest should be noted; the rate after a simple effort test should also be determined. Such a test might consist in walking about 50 feet or climbing a few steps; the practitioner will devise such tests within the limitations and facilities of his own office. After average standards have been established from the results of normal subjects of various age, weight, and sex groups,

distinct systolic bruit on auscultation and occasionally a thrill may be detected.

Pulsations over the anterior chest are common; in the precordial area the apex impulse is frequently extensive. There may be pulsations in adhesive pericarditis and sometimes in pleural-pericardial adhesion syndromes. Coarctation of the aorta may also produce thoracic pulsations; these may be posterior. Arteriovenous aneurysms are not too common in general practice; most of these are traumatic or infectious in origin and the most common site in the author's military experience was in the legs, although, a few arms were also involved.

Inspection of the venous system should not be neglected; since increase in venous pressure is the rule in heart failure, there is usually an increase in the superficial venous network, especially in the dependent parts of the body. Note should be made in regard to the extent and degree of venous engorgement. With the patient in the standing position, competency of the valves should be determined and in the reclining position, with the legs held high, emptying time should be noted. Superficial and deep thrombosis, if present, should be studied for possible infection. Pulmonary embolism from venous thrombosis is not infrequent in congestive failure

Finally, inspection of the feet is important in cardiovascular disease, [illegible]
eral vascular con[illegible]
Small cuts and abrasions in congestive failure with edema of the feet and lower legs may lead to intractable infections which seriously interfere with convalescence; ulcers are notoriously difficult to handle under such conditions.

clothing within the demands of modesty; no heart can be examined through a small opening in a man's shirt or by slipping a stethoscope under a tight brassiere or girdle. Women should be covered with an acceptable gown; men may be examined in shorts. Genital examination is rarely necessary in cardiology; a rectal inspection may be required to establish the presence of hemorrhoids in some cases of congestive failure. Nearly all patients will cooperate if they are told that, while the heart is in the chest, the blood vessel system extends throughout the body and every part has to be inspected for abnormalities.

Unusual pulsations of arteries should be noted; most of these occur in the neck. It is seen in simple hypertension, *in aortic insufficiency, and in aneurysms of the aorta* or great vessels of the neck. Abnormal pulsations are also noted in the axilla and upper arms as well as in the anterior cubital space. The inguinal and posterior popliteal areas are also common places for the discovery of abnormal arterial pulsations. Isolated pulsations have, in general, more clinical significance than generalized; the latter, as indicated before, is usually due to aortic valvular insufficiency or hypertension. Isolated pulsations are more likely to be due to aneurysmal changes in the local artery. Aneurysmal dilatations must be differentiated from tumor masses overlying an artery; the latter may appear to be pulsating but palpation will usually indicate the mechanism. Such tumor masses may be large inguinal or axillary glands; a so-called pulsating thyroid may be due to motion transmitted to a normal gland from an adjacent or underlying aneurysm. Many pulsating aneurysms produce a

cardiac conditions; the rate may start at 80, rise to 120, and drop to 50 when the respiratory cycle is slowly and forcibly performed. Premature beats may develop during the expiratory phase when the rate is slow; electrocardiographic study is indicated in such instances to determine the origin of the extrasystole. This will be discussed later in relation to electrocardiographic interpretation. No change in rate or rhythm during the respiratory test is common in elderly subjects, this loss of vagal response may have clinical implications in regard to exercise. A similar loss of vagal response is noted in several types of cardiac disability, more especially when failure develops. If irregularities are noted during auscultation a presumptive diagnosis should be attempted; the challenge offered should not be neglected for there is presented here a unique opportunity to put experience and deductive reasoning against technologic examination by the electrocardiograph or other apparatus. By this method the practitioner will quickly increase his diagnostic acumen and self confidence in the presence of irregularities which formerly appeared to be baffling disturbances.

The rate of the heart beat should be noted under several circumstances. The normal, well conditioned heart shows relatively little change, standing, sitting, or in the recumbent position, there may be little or no change after the simple effort test. For example, the pulse rate of a well known athlete was 74 standing, 72 sitting, and 72 recumbent; after 50 walking steps it was 74. This can be compared to a man of the same age, 32, with moderate congestive failure from valvular heart disease; here the standing rate was 88, sitting 80, recumbent 74, after the

Auscultation

Study of the heart sounds has already been discussed in Chapter I; the Alsahy work sheet provides spaces for notes in regard to the two sounds at the apex as well as at the base. The characteristic of quality and intensity or loudness should be estimated; space is provided for a description of unusual auscultation phenomena like reduplicated or split sounds or an accentuated third heart sound. Regardless of whether a coding system is used, it is advisable to fill out completely the estimated loudness and quality of the four special heart sounds; this will be used on subsequent examinations to determine changes in the first and second sound at the apex and the second aortic and second pulmonic heart sounds at the base. It should be repeated that too much time cannot be spent in auscultation; the neglected time factor in heart sound examination is usually the time spent in listening.

Rhythm of the heart beat can well be studied during this extended time factor; it may require 3 minutes or longer to determine the presence of an occasional extrasystole or the missed beat of second degree heart block. Short runs of atrial fibrillation or flutter may be missed as well as *paroxysmal tachycardia. The effects of vagal stimulation* should be determined in every patient; the simplest test is the respiratory response in rate and rhythm. Normally, deep inspiration is accompanied by an increase in rate and forced expiration with a slowing; this is the usual rhythm in children and young adults. Sinus arrhythmia may, however, appear in exaggerated form in certain

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systolic in time; they occur in the interval between the first and second heart sound. In the coding system previously described, this interval is divided into three equal parts which are known as early, mid and late systole. When the rate is slow, it may only require moderate concentration to identify in which part the murmur reaches its maximum intensity or loudness. Many systolic murmurs have no clinical significance; in general, those murmurs which fill the entire systolic interval are the most important. The systolic murmur of mitral insufficiency and that due to aortic valvular stenosis usually fill the systolic interval with equal intensity in all three parts. The systolic murmurs heard in congenital heart disease also occupy the entire period. Functional systolic murmurs ordinarily fill only the first or second parts of the interval; functional mitral murmurs, as the name implies, are not due to anatomic changes in the mitral valve mechanism. Relaxation of the ring, due to poor muscle fitness from one cause or another, results in an orifice which is greater in diameter than the spread of the valve leaflets; such functional murmurs show many changes in tonal quality and intensity depending upon complete or partial restoration of the orificial ring structure. Increasing heart muscle tonus frequently permits the ring to assume its normal size with a return of valvular competence. In some instances, deep respiratory exercises may be sufficient to produce this result and the murmur may entirely disappear; in others, change in position of the body may cause alteration in the murmur. After exercise with an improvement in muscle tonus, the murmur may disappear only to return again after a resting period. The transmission of the murmur

walking test 112. Provided sinus rhythm is present, it is a general rule that the greater the disability the greater will be the spread between the 4 standard pulse rates.

In normal sinus rhythm, the rate at the apex of the heart and the rate at the radial artery in the wrist are identical; if premature beats occur there may be a pulse deficit. In other words, there will be more apical beats than radial beats if the extrasystole is very permature; this mechanism is explained elsewhere. Both the rate at the apex and at the wrist should be recorded in all irregularities. In atrial fibrillation, for example, the radial rate alone may give entirely false information; there may be a great pulse deficit with an apical rate of 140 and a radial of 80. In hospitalized patients or, where a nurse is employed for a given case, specific instructions should be given that both rates are to be determined; where the pulse rate at the wrist is a routine procedure, no accurate information concerning the actual rate of the heart can be arrived at from the radial rate alone. The same is true of office practice, if a nursing assistant is assigned the duty of determining the pulse rate.

Murmurs

The discovery and studying of murmurs is oftentimes considered to be the *sine qua non* of auscultation. Until the characteristics of the heart sounds have been definitely established, the identification of murmurs is not possible. Timing in the cardiac cycle and the relationship of the sound changes are necessary to evaluate the significance of any given murmur. The most common murmurs are

normal predominance. At the base of the heart, the normal sound pattern shows the second aortic predominating over the second pulmonic in nearly all adults; in mitral valvular disease, a reversal of this sound pattern also occurs. The second pulmonic is usually markedly accentuated in mitral stenosis as well as in any syndrome in which there is an increase in pressure within the pulmonary circulation. In long standing cases of mitral insufficiency, the second pulmonic becomes louder than the second aortic; in less advanced instances both the second aortic and second pulmonic may have the same tonal intensity; the explanation here is that the second aortic has not lost its normal characteristics but the second pulmonic has increased in loudness and duration.

A more definitive approach towards the differential diagnosis of functional and organic systolic murmurs is thus made possible by the utilization of these observations. It has long been recognized that one of the most difficult problems in clinical medicine is such differentiation; no method has received universal acceptance and there is no intent here to suggest that changes in the heart sound pattern is the final answer. But in any given case in which the systolic murmur (1) does not fill the entire systolic interval, (2) shows changes after the expiratory, postural, and exercise tests, (3) is not accompanied by accentuation of the first sound at the apex, (4) has an increase in the loudness of the second pulmonic sound, the evidence indicates that it is functional. It is difficult to state whether more harm has been done over the years in considering functional murmurs organic or organic murmurs to be functional; in the first instance, individuals have been

toward the axilla, which formerly was considered to be of differential diagnostic importance, has been found to be of little significance; experimental phonocardiographic studies have shown that both types of systolic murmurs may show similar transmission characteristics.

Early and midsystolic murmurs heard with maximum intensity over the pulmonic valvular area are usually functional; the organic systolic murmur of pulmonic artery stenosis fills the entire systolic interval. Functional systolic murmurs rearely occur over the aortic valve; here the systolic bruit due to atheromatous or sclerotic changes of the valve mechanism or to the first portion of the aorta must be distinguished from a true murmur. Like the functional systolic murmur, systolic bruits tend to change tonal characteristics with alterations in blood pressure and blood velocity. After exercise such systolic bruits usually become louder and have a high pitch; they do not disappear but when the patient is quiet or asleep they may become very difficult to hear.

Systolic murmurs due to valvular pathology are usually associated with heart sound changes. In the presentation of the coding system in Chapter I, it was pointed out that the normal heart sound pattern in the adult shows the second sound at the apex to be louder and longer than the first; the second sound was said to predominate. In mitral valvular disease, the first sound tends to predominate; this reversal of the normal sound pattern is characteristic in advanced mitral stenosis, where the first sound is traditionally spoken of as "loud and snapping." The predominance of the first sound is less marked in mitral insufficiency but even in early cases the second sound loses its

time in relation to systole of the ventricles; it is during this point in the cardiac cycle that the atria are contracting. The murmur of mitral stenosis is thus late diastolic or presystolic in time and it occurs before the first heart sound. While these facts are well established, it may be difficult in certain instances to correlate the sounds, murmurs, and timing factors into a single clinical syndrome. Murmurs may be so loud that the sounds cannot be distinguished or the sounds may be so accentuated as to obscure the murmurs; when irregularities are present, as in rapid atrial fibrillation, the condition has been aptly described by Parsonnet as "auscultatory chaos." Yet, from such a cacophony of sounds, a symphony of clinical analysis and interpretive understanding should be possible in nearly every case.

The first step in the analysis of the heart sounds in any cardiosonic examination is the identification of the first and second sounds at the apex; in sinus rhythm no great problem is presented although splitting of the first or second sound may sometimes confuse the sequential relationship. In gross irregularities of rhythm, however, like paroxysmal atrial fibrillation, with or without flutter, and paroxysmal nodal and ventricular tachycardia, the rate may be so rapid that differentiation is impossible. In rates of 160 and over only one sound may be noted; this sound is usually the first. The dying heart noted pre-agonally in many diseases with its associated poor, rapid, and thready pulse, frequently shows the "one heart sound syndrome"; the loss of the second sound carries a generally guarded outlook since this is a hemodynamic phenomenon of a failing left ventricular output. Thus, in very rapid

forced to carry a cardiac diagnosis with all its implications throughout their social, economic, industrial, and perhaps sexual life and in the second misdiagnosis, patients may have suffered an increase in their disability because of the freedom of activity offered by the belief that they had a normal heart. There is, thus, a tremendous burden of clinical responsibility which the practitioner must face in every case where the mechanism of the systolic murmur is in doubt.

Fortunately there are other diagnostic methods which may contribute material data; the size and shape of the heart determined by X-ray, the electrocardiogram in certain cases, and finally catheterization and contrast media studies are available. In the vast majority of cases, however, auscultation remains the chief method of making the differential diagnosis and the experienced clinician is able to reduce the margin of error by painstaking study of the heart sounds and murmurs.

Diastolic murmurs are timed between the second and the next first heart sound; since the diastolic interval is much longer than the systolic, the division of the interval is much easier to make. Diastolic murmurs are much less frequent than systolic; the ratio in large cardiac clinics is about 1:10. The predominance of systolic murmurs is due, in no little extent, to the addition of functional murmurs in the statistical data; there are probably no functional diastolic murmurs. Diastolic murmurs arising as the result of aortic valvular incompetence may fill the early, mid, and late parts of the interval either alone or in any combination; in advanced cases the entire diastolic interval is occupied. The late division of the interval is presystolic in

ure gradient changes during atrial and ventricular systole, are but a few of the known factors which together are responsible for auscultatory phenomenon recognized as a cardiac murmur. In mitral insufficiency, the intensity of the murmur may bear no relation to the actual incompetency of the valve; the volume of blood forced back into the left atrium can not be estimated by the pitch, intensity, or duration of the systolic murmur.

Likewise, the tonal characteristics of the presystolic murmur of mitral stenosis with its classical crescendo quality, is apparently not so much due to the actual narrowing of the orifice as to the shape and contour of the opening and to the loss of resiliency of the tissue structures. Rigid fibrous changes with more or less extensive calcification, which produce a slit-like valve orifice of the so-called button hole group, are more likely to show the loud crescendo type of presystolic murmur than the circular or oval form of stenosis. Here, too, the characteristics of the murmur show no consistent relationship with the degree of valvular obstruction; narrowing of the valve leads to a rise in left atrial pressure which in turn increases pulmonary pressure, first on the venous side and then arterial. Catheterization of the pulmonary artery and the determination of "wedge pressure" in various types of mitral stenosis has shown that the greatest increase in pressure is not necessarily associated with the classical type of presystolic crescendo murmur.

Mitral valvular murmurs have thus lost much of their former clinical significance insofar as their various characteristics have been employed as a measurement of a given pathologic process. The coding system still retains a

rhythms, if only one sound can be definitely identified, this can be assumed to the first sound and all other auscultatory findings may be interpreted in relation to their position with a point in the cardiac cycle which ends in the diastolic period and which starts in the systolic. If a murmur follows this single sound, it will be systolic in time; if it precedes the single sound, it will be diastolic but which type may not be clear.

Determination of the specific irregularity is more than a simple contest of skill and wit, although the challenge has a certain sporting aspect among the intern staff members of every hospital before the electrocardiographic examination is made. Treatment and to some extent prognosis, will be based upon the predominating etiological background for the abnormal rhythm; accurate diagnosis may spell life or death in some cases.

Murmurs as well as heart sounds may be graded in terms of intensity of loudness; in the Alsahy coding system, all murmurs have a 1 to 5 scale of auscultatory intensity. Before the advent of open cardiac surgery, it was generally assumed that a grade 4 systolic murmur, for example, was produced by a more extensive pathologic process in the mitral valvular structure than that associated with a grade 2 murmur. Thus, a grade 4 systolic murmur was traditionally considered to be more advanced and to have a more serious outlook than a grade 2 murmur. The mechanism of murmur production is apparently far more complicated than the actual widening or narrowing of a valve orifice; length of the traveling distance of the blood stream through a distorted valve, angulation and current eddies, counter velocity streams from recoil action, pres-

valve orifice, are very similar in their auscultatory pattern as the grade 4 and 5 diastolic murmurs of luetic origin with its descending aortic pathology and rolling of the valve leaflets into a rigid oval ring. Changes in the grading of diastolic murmurs is very less frequent than with the mitral murmurs, except in acute rheumatic involvement; in children and young adults the alteration is more likely to be in duration rather than in intensity. An early and mid-diastolic murmur may in the course of a few days develop into a complete diastolic interval murmur; this change can readily be misinterpreted as an increase in *loudness* rather than in length.

At the *initial* examination of the patient, all murmurs should be coded for future reference. The coding also serves as supplementary evidence in establishing a diagnosis when considered with the data obtained in the study of the heart sound characteristics. The complete formula represents a clinical appraisal of stethoscopic examination which may play an important part in the final disposition of the case.

Pulmonary Examination

The lungs are a part of the cardiovascular system but diseases of the lungs *per se* are, in the present subdivision of medicine, a specialty almost cut off from cardiology. All diseases of the lungs, in lesser or greater degree, involve the vascular component structures while cardiac disturbance, particularly of the right heart, has many pulmonary implications. The syndrome of cor pulmonale is an example of combined pathology of both systems. In

high degree of usefulness in recording the temporal aspects of valvular involvement by certain disease processes. In acute rheumatic fever, for example, tonal alterations of the murmurs may occur from day to day; a grade 2 systolic murmur may increase to grade 3 or 4 and then subside in a few days or weeks to grade 1 or 2. Such changes in the grading classification may be taken as clinical evidence of active infection and as such may be utilized for specific treatment. Systolic mitral murmurs which develop in the course of an acute coronary episode may be viewed in the same manner; here, the pathology is apparently associated with myocardial changes taking place in the muscle bands which are included in the structural complex of the superficial and deep valve area. These systolic murmurs may change from day to day and sometimes from hour to hour in massive infarction; they must be distinguished from the systolic phase of a pericardial friction rub which may develop under the same circumstances. In the typical rub, the systolic phase is nearly always louder and better localized than the diastolic; the latter may be so diffused that it is heard with great difficulty. If it is missed or unsuspected, the systolic component of the friction rub may easily be mistaken for a systolic murmur.

The diastolic murmur of aortic valvular insufficiency has not received as much surgical attention as the mitral murmurs; the correlation studies of murmur characteristics and incompetence of the valve have as yet not provided informative data of any considerable clinical value. The very loud grade 4 and 5 diastolic murmurs, seen in advanced rheumatic diseases with triangulation of the

logic methods of examination and it is true that the X-ray is a simple way out of a doubtful diagnostic thoracic problem. But percussion still remains a simple method of determining certain changes in the thorax which are of significance in heart disease. Flatness at one or both bases is common in congestive failure and, if discovered, will set a treatment pattern; the rise or fall of the flatness level may be an index of therapeutic control. The percussion should be marked and left permanently upon the chest wall; Cabot was fond of saying that a well marked chest was a well treated patient. Daily X-rays may not be available but daily percussion should be routine. It should be pointed out that the flatness level does not necessarily represent a fluid level; flatness is also a phenomenon of lung compression, especially at the bases. A relatively small amount of fluid may produce a relatively higher flatness level than that suggested by the actual hydrothorax. The place of diagnostic and therapeutic thoracenthesis will be discussed later but the discovery of basal or other flatness has many clinical implications.

Auscultation of the lungs, insofar as heart disease is concerned, is chiefly focussed upon the discovery of rales produced by the failing heart. Here, the skill and experience of the examiner stands alone; rales can not be photographed upon an X-ray film and the stethoscope is not challenged by any other instrument or laboratory method. Various types of rales are described by authors interested in the pulmonary diseases and sound recordings are available for the differential tonal and timing characteristics of the common rales; the practitioner will profit by obtaining such phonograph records or tape recordings. Sound rec-

this presentation, only the cardiovascular aspects of pulmonary examination are emphasized. This is, perhaps, unfortunate for it represents the triumph of expediency over scientific medicine; it is one of the penalties of specialization set by the scope of practical cardiology.

Examination of the chest as a whole is prompted for two compelling purposes; the first is in relation to cardiac function and the second concerns the structural environment in which the heart performs its work. Abnormal changes in the size, position, and physiologic characteristics of these structures in a closely packed thoracic cavity will alter the functional capacity of the normal heart and great vessels; if the latter are already burdened by pathologic processes, the degree of cardiovascular impairment may be markedly enhanced.

Auscultation of the chest thus provides both actual and presumptive data of considerable clinical importance. Physical examination, here, starts with inspection of the thoracic respiratory movements; unequal or absent boney cage changes as well as intercostal muscle alterations should be noted. Palpation for thrills or other vibratory phenomena may be revealing; the finger tips of the examiner may be less sensitive in certain cases than the open palm of the hand held firmly against the chest wall. All parts of the chest should be so tested for abnormal vibrations with particular attention to the anterior surface; in women, the areas *under* the breast should not be overlooked. Superficial voice transmission and the cough impulses are also informative.

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ords of heart sounds, murmurs, and pulmonary sounds will have increasing space in the medical bookshelf of future doctors; no book can be as eloquent in describing the characteristics of sound than listening to the sound itself. The rales produced by early congestive failure and described as fine or crepitant have been liken to the crisp crinkling of thin paper in contrast to the heavy coarse rales noted in pneumonia, bronchial asthma, emphysema, and other lung conditions. Nearly all rales are diphasic; those produced by expiration are usually louder than those of the inspiratory phase. While no general rule can be set down, the early rales of congestive failure are usually more evident during the inspiratory phase.

Auscultation just above the percussed level of flatness, if this is present, may show these rales in abundance but in congestive failure all parts of the lung may show crepitation. Coughing does not remove these rales which arise in the alveoli and finer subdivisions of the lung structure; rales which have their origin in the medium and larger bronchi may disappear or lessen after coughing. Auscultation of the areas of flatness frequently reveal "tinkling water sounds" which are best heard during the deep inspiration with fall of the diaphragm. This water drop tinkling or subminimal gurgling may follow the fluid collection during the postural test of hydrothorax; in many instances the areas of flatness does not move appreciably as, for example, when the patient changes from a standing to a bending forward position even though most of the fluid is more or less free. This water drop sound pattern is more common in the right chest, due, perhaps, to the greater number of interlobar spaces.

Relatively large amounts of fluid may accumulate in the thorax before an increase in respiratory rate occurs; this is somewhat paradoxical since vital capacity diminishes rapidly with the onset of congestive failure. There is, however, a close correlation between the discovery of rales and pulmonary function and the determination of vital capacity may be confirmatory of myocardial failure when few other signs are obvious.

Rales may develop early in mitral stenosis if there is a relatively rapid increase in pulmonary pressure; in the carefully controlled exercise tolerance test, crepitant rales may be discovered within a short time and tend to disappear in about 20 to 30 minutes. When such rales occur there is a marked drop in the Cardio-Pulmonary Index as well as the Respiratory Index. As a test of cardiac functional capacity in mitral stenosis, the appearance of such rales has a number of clinical implications, more especially if they are not present during the patient's normal activities.

Examination of the Abdomen

The onset of congestive heart failure may show its first objective evidence in the abdomen. In a large series of patients entering New York City Hospital with heart disease and edema, a statistical study was made of the apparent initial localization of fluid retention. It was found that swelling of the ankles and lower legs lead the list, followed by abdominal changes, and pulmonary involvement was least; perhaps, edema of the ankles is more easily observed by the patient but a comparative investiga-

tion made among clinic physicians and other practitioners produced a list of similar relative appearances of the signs of congestive failure. In any given patient, however, any of the three major areas may be the first and may remain the only obvious focus of edema. Experimental physiologists, like Opitz and Plungian, were unable to offer a definitive concept concerning the localization of the edematous process in heart disease; it has been well recognized for over a century that no prediction can be made concerning the probable first appearance of fluid retention in a cardiac patient. Many individuals who experience heart failure over long periods of time, only show manifestations of edema in one area; a patient, for example, with marked liver and spleen enlargement and ascites may have little or no dependent edema and no pulmonary signs. Likewise, the pulmonary area may bear the brunt of the pathology with few signs elsewhere. Indeed, hydrothorax may be confined entirely to one side. In advanced stages of congestive failure all areas may be involved but, even under these extreme circumstances, one area still retains a predominating position. Moreover, patients with a history of many bouts of failure, usually show a repeated pattern of fluid localization.

Examination of the abdomen thus presents an opportunity to determine the objective signs of edema due to the failing heart. The liver should be carefully palpated; many authors have described methods of examining the liver. Of them all, the Wolffe procedure is perhaps the most useful. The examiner takes a position facing the patient's feet, not in the usual manner of facing the head of the bed or table. With both hands gently but firmly placed just under

the costal margin, the subject is told to make a long expiration and hold it; the fingers are then inserted as far under the rib margin as possible. The patient is then instructed to inspire slowly and deeply; as the diaphragm descends, the liver edge will tend to slip over the fingers. In early hepatomegaly and in borderline enlargement, the method may be rewarding. Obvious edema of the liver requires no special skill in palpation; the patient himself may state that his liver is enlarged or is getting bigger.

The liver in congestive failure is usually symmetrically enlarged; it is rare to have only one lobe involved. The surface is generally smooth and the edges distinct but rounded. In the beginning, the swollen liver may be very tender to touch but in chronic enlargement there is little or no painful reaction to deep pressure. Increase in abdominal girth may occur but in previously obese subjects no obvious enlargement in waist measurement may be noted either by the patient or family even when gross hepatomegaly is present. The large liver in congestive failure may be singularly free of subjective symptoms; a feeling of abdominal heaviness is usually described but, within the individual's limited capacity for activity, there is relatively minor discomfiture. This stands in contrast to other causes of hepatomegaly like cirrhosis, metastatic tumors, and certain infections. Icterus is a rather late symptom in congestive enlargement of the liver although it was described as an early finding by many authors at the turn of the century; this may be due to the better methods of handling congestive heart disease since the discovery of mercurial and other diuretics as well as more definitive measures in treatment. When jaundice occurs, it

usually is of a mild type which may be missed on inspection of the patient's skin; examination of the sclera is a more reliable guide of icterus. Laboratory tests are, of course, indicated in any instance of suspected jaundice.

Palpation of the spleen should be a routine procedure; in advanced congestive failure there may be considerable enlargement but, as a rule, splenomegaly is a rather late objective finding compared to liver enlargement. The swollen spleen is far more sensitive in relation to the patient's own reaction to its enlargement. Discovery of splenic enlargement may be difficult; it may be noted sometimes on change of position. With the patient lying on the left side, or face down, the respiratory tests may bring the edge within the finger tips. In many instances of congestive failure with enlarged liver, there may be no associated splenomegaly even on post mortem examination but in many cases marked enlargement of the spleen has been discovered at autopsy which defied detection on physical examination. Identification of a swollen spleen is a positive finding but its absence does not necessarily indicate that it is not present as a part of the congestive failure syndrome.

The demonstration of free fluid is not always a simple procedure even when large accumulations are present; ascites in patients with large obese abdomens may only be a post mortem finding. A recent case was a 58 year old man with a long history of hypertensive heart failure with 6 separate bouts of congestion; he was a large individual, weighing about 225 pounds at his calculated dry weight. He had a generalized anasarca of his legs and lower abdomen, a large liver, but his lungs were more or less clear.

Ascites could never be determined on repeated examination He died during an episode of ventricular fibrillation. Autopsy showed over 12,000 cc. of ascitic fluid. Another patient, a 44 year old woman with mitral stenosis, atrial fibrillation, and marked congestive failure had repeated abdominal taps in which 2 to 3 liters of fluid were removed; a paracentesis had been performed about 4 hours before death and all fluid was presumably removed. At post mortem an additional 4000 cc. was measured. Estimation of free fluid in the abdomen is always more difficult than in hydrothorax where percussion and X-ray present specific data. In the abdomen there are many possible pockets for fluid collections; "free fluid" may have a relative connotation. The importance of diagnosis stems chiefly from the therapeutic need of its removal; in addition to the usual vagaries of fluid accumulation in other parts of the body in congestive heart failure, the development of ascites has many distinct characteristics of its own. The first is that there is no correlation with either hepatomegaly or splenomegaly and the appearance of free fluid; minor liver enlargement may be accompanied by considerable fluid. The reverse is equally true. The second clinical experience is that no further ascites may develop after a single tap while, in other cases, one tap may usher in a long series of paracentesis procedures. Electrolyte and water balance is discussed elsewhere but it may be pointed out that there may be a close relationship between electrolyte metabolism and ascites development and the removal of much fluid may be responsible in part for its re-accumulation. Many cardiologists doubt the wisdom, therefore, of complete removal of ascitic fluid in

certain patients. Unlike hydrothorax, fluid appears to be readily adsorbed from the abdominal cavity and during therapeutic diuresis large quantities may disappear.

Examination of the Lower Extremities

The general physical examination concludes with inspection and palpation of the legs. It has already been indicated that edema of the ankles was the most common site of fluid collection in congestive failure. Patients, particularly women, are more likely to notice swollen ankles or feet than any other early sign of the failing heart; it may take precedence over dyspnea, precordial pain, or palpitation. When an otherwise normal looking ankle in the morning, gives way to thickness at night when shoes are removed, the symptom always carries significant forebodings. Men react less to the changed appearance at night compared to the morning size but the finding is not generally ignored. On the other hand, some patients accept swollen ankles and legs with apparent unconcern and perhaps with the mistaken notion that they are naturally gaining weight as they grow older. This idea is sponsored in some measure by the fact that they are weighing more actually and it should be recalled to the unconvinced individual that each pound so gained may represent the retention of one pint of fluid which has no functional use in the body and must be removed.

Early pitting edema is not always easy to demonstrate, particularly in men with thick skinned, heavy ankles, here edema of the dorsum of the foot may be the diagnostic site. Pitting edema of congestive failure must be differen-

tiated from swelling due to other conditions; localized areas of edema from venous obstruction (varices), orthopedic problems (traumatic and infectious), skin infections (fungus), endocrine syndromes (myxedema), avitaminosis (beriberi), malnourishment (starvation edema) are but a few of the more common clinical conditions which will be seen by the general practitioner. It is not impossible for any one of these alone or in combination to be found in cardiac patients; this is true of neglected patients who have no medical care for one reason or another. Such patients are not uncommonly seen in cardiac clinics serving city hospitals in large communities.

Unilateral pitting edema may pose some interesting diagnostic questions; the condition is not infrequent in early congestive failure. For the same unpredictable reason previously considered in relation to the localizing site of edema in heart failure, one ankle instead of both may temporarily be the focus of the impaired circulation. Where there is evidence of superficial or deep venous disturbance, forward myocardial failure may be aggravated by dysfunctional return flow to the right side of the heart; unilateral ankle edema could occur under such circumstances but some patients with one-sided edema show no objective signs of venous pathology. The necessary proof of its cardiac origin may require a test of therapeutic response. A 56 year old woman college librarian noted a slight swelling of her left ankle which was more evident at night; she had no local pain or disability. Orthopedic and X-ray examinations were negative; a tight ankle bandage caused swelling to appear above the usual area of pitting edema. In the course of a general physical examina-

tion, a mitral systolic murmur was noted but the patient had been aware of this condition for many years; she confessed reluctantly to limitation of activity due to dyspnea and palpitation. A short course of digitoxin, aminophyllin, and diamox improved her subjective symptoms and the unilateral edema of the ankle completely disappeared. Not all such cases are so responsive and bilateral ankle edema may subsequently develop even with specific treatment.

Massive edema involving both legs to the thighs and above, ordinarily present no diagnostic problems in this country; a few of the South Pacific infestations, like filiriasis, have been carried back by servicemen and such individuals have been seen from time to time in Veterans Administration Hospitals. Former G.I.s, who have been in areas where parasitic infestation was endemic, should be questioned; the practitioner may occasionally discover such a case. Such individuals usually show no cardiac symptoms; the pathology is one involving the lymph and venous systems of the legs and sometimes the scrotum. Massive anasarca from congestive failure can also produce scrotal edema; the skin of the lower abdomen and deeper layers may show a marked pitting edema. Most of these patients are seen in the neglected group previously mentioned; it is a rare case under modern management that advances to such a state of decompensation.

Examination of the legs should include a note concerning the palpation of the dorsalis pedis artery and the posterior tibial if possible. Superficial venous pressure tests utilizing the leg elevation procedures should be performed and color changes of the feet, if present, should be re-

corded. The pigmented scars of old, healed varicose ulcers should also be noted as well as the occurrence of ecchymotic spots. The general circulation of the toes in relation to color, temperature, and freedom from fungus infections, require a few words which subsequently may have considerable clinical importance.

Blood Pressure

The determination of blood pressure like the examination of heart sounds and murmurs requires more time than the superficial approach which is sometimes given to this important study of cardiovascular function. The physiologic factors responsible for both systolic and diastolic blood pressure are among the most complex mechanisms in the body; some of these are known but it is probable that many important factors remain to be discovered. Systolic pressure is predicated upon a number of compensatory adjustments, which at any given instant, are constantly changing. Experimental studies show that the absolute pressure gradient varies from beat to beat; changes in ventricular output, blood velocity, time factors, arterial capacity, local and peripheral resistances, neurogenic and psychologic influences, humoral effects, are only a few of the recognized physiologic phenomena involved in the production of blood pressure. Any one factor may predominate as well as any combination; systolic pressure is thus a very dynamic clinical finding and the figures which are obtained by any given apparatus are only as valuable as these factors are understood.

Every practitioner is familiar with blood pressure de-

termination and with the various types of apparatus in use, but a few words may be permitted in regard to some of the errors which may occur in the casual and superficial methods occasionally employed. The accepted procedure recommended by the combined committee of the American Heart Association and its British counterpart emphasize that clinical systolic pressure is that point in deflation of the arm cuff when vascular sounds are first heard. If the heart rate is slow and the drop in the cuff pressure is rapid, the difference between one beat and another may represent a difference as much as 20 mm. Hg.; the faster the rate and the slower the deflation, the more accurate the observation is likely to be. Insofar as 10 mm. Hg. may spell the difference between an accepted normal and an elevated blood pressure in the critical range from 140 to 150, an accurate estimation requires careful observation of the first auscultatory sound. Many factors are involved in this sound, and not the least of which, are the examiner's own problems of hearing and seeing. The patient's psychogenic response to the examiner and to the examination play an equally important part in the determination. As an object lesson, the author has frequently invited a number of physicians and nurses to take a given subject's blood pressure; a variation in systolic pressure from 10 to 25% is not uncommon. Diastolic pressure shows fewer variations in spite of the difficulty in determining the end point, a factor which will be discussed later.

On the initial examination of the patient, blood pressure should be taken on both arms; a note in regard to *handedness* should be made. Systolic pressure has a tend-

ency to be somewhat higher on the side instinctively used; thus, in right-handed subjects who are physically fit, the right arm pressure may normally be 10 to 20 mm. Hg. higher than the left; the reverse is equally true in left-handed individuals. The question which pressure should be accepted, naturally arises. Most cardiologists believe that the higher level has wider practical implications. Marked differences in the two arm pressures, however, suggest certain congenital or acquired lesions, the chief of which is coarctation of the aorta.

Blood pressure should be taken in several positions and after the simple effort test. These determinations may be expedited if an established routine is maintained. The initial pressure of both arms is taken first in the standing position; if no great variation is noted, it may not be necessary to repeat the double arm examination again, except in indicated conditions. Subsequently, either arm can be used, but certain tests may require consistency in the employment of one arm. After the patient has been lying down for several minutes and during which time the abdominal and leg examinations have been made, blood pressure is again determined. In normal subjects, this pressure usually is lower than in standing, but patients with cardiovascular disease may show an elevation. The individual is then instructed to stand up quickly; in normal subjects there is a rise in systolic pressure. In heart disease, there may be a marked fall; this type of postural hypotension has been previously described. A drop of 20 to 30 mm. Hg. is not uncommon and may be accompanied by considerable vertigo and unsteadiness. After the simple effort test, there may be a moderate rise in pressure with

a return to the original standing levels within 5 minutes.

In hypertensive syndromes two general pattern curves are found when this procedure is followed; in the first, and most common, there will be marked changes from one position to another with a variation range of 30 to 60 mm. Hg. For example, a 42 year old lawyer showed a standing pressure of 188/110; recumbent 196/106; quick standing 156/100; after simple effort 222/116; after 5 minutes resting 180/100. This curve stands in contrast with another 44 year old man with a standing pressure of 190/106; recumbent 192/108; quick standing 190/108; after effort 200/112; after the 5 minute rest period 190/106. Hypertension with a fixed and unchanging function curve has a more unfavorable outlook than the so-called flexible type; the latter is definitely more responsive to therapy and other accepted measures. In many instances, the practitioner may be able to predict with some accuracy which of the two groups is more likely to benefit from treatment.

A word about diastolic blood pressure and its determination; the errors which creep into routine systolic level estimations are even more prevalent here. Where the end point of the last vascular sound is clear, distinct, and definite, there may be no problem. But even in normal subjects there may be 2, 3, or 4 diastolic end points which may vary as much as 5 to 15 mm. Hg. In the critical level of 100 mm., the difference between 95 and 110 mm. Hg. may have a diagnostic significance out of proportion to the actual facts. When the diastolic end point is questionable and difficult to fix with an accuracy which a given case may require, it may be well to recall that the so-called reverse method of Jacobi, described in 1910, may

be of value. Diastolic pressure is determined on a falling pressure gradient; that is to say, the arm cuff pressure is slowly decreased until the vascular sounds disappear. In the Jacobi method, the cuff pressure is slowly raised from 0 mm. Hg. until the first arterial sounds are heard. The method is more difficult to perform because the noise made by contractions of the hand bulb and the arm cuff as it becomes inflated seriously interferes with the less easily heard vascular sounds. However, with a little practice the reverse method may offer confirmatory data concerning the true diastolic level.

Diastolic pressure is far less mobile than systolic; whereas the systolic levels may exhibit a wide range in the different positions as previously noted, diastolic pressure usually does not differ more than 5 to 10 mm. Hg. The normal level of diastolic pressure in physically fit subjects is usually below 90 mm. Hg.; the average diastolic pressure in normal individuals in large urban areas is 10 mm. Hg. higher according to several recent surveys. From a clinical point of view, the critical level of diastolic pressure carries a far greater diagnosis and prognostic responsibility than the critical systolic level. The practitioner must evaluate all of the circumstances attending a patient with a border line diastolic pressure; the importance of individualizing each case cannot be overemphasized for it is in this group that a number of problems may subsequently develop as the result, perhaps, of misdiagnosis.

Pulse pressure assumes clinical importance as a measure of acute myocardial failure. Pulse pressure is the mathematical difference between systolic and diastolic blood pressures; thus, a given pressure of 130/80 presents a

pulse pressure of 130 − 80 = 50 mm. Hg. When pulse pressure falls to 20 mm. Hg. and below, those physiologic functions which are dependent upon pressure gradient factors become disturbed and may cease their specific activity. The kidney, for example, requires a minimal pulse pressure of 15 to 18 mm. Hg. to perform its several intrinsic activities; the liver ceases to function at 10 to 15 mm. and certain portions of the brain associated with autonomic nervous system control also show serious dysfunction at 15 to 20 mm. In acute coronary artery occlusion with myocardial infarction, pulse pressure usually falls; if shock is present pulse pressure may be as low as 10 to 20 mm. The intra-arterial injection concept of such shock therapy is based on the attempt to increase pulse pressure. On the other hand, low pulse pressure is also a hazard in acute pulmonary edema due to mitral stenosis; here the effects of increased venous pressure are added to poor left ventricular output. A complex hemodynamic situation is presented; venesection is indicated to relieve right heart strain. With removal of venous blood, pulse pressure usually improves. In the instance of myocardial infarction shock, however, subminimal pulse pressure may be benefited by intra-arterial blood injection, while in acute pulmonary edema removal of venous blood may serve the same function.

Average blood pressure is the simple addition of systolic and diastolic pressures divided by 2. Thus, a blood pressure of 130/80 will give an average pressure of 105;

$$\left(\frac{130 + 80}{2} = 105\right).$$

The determination of average blood pressure has not been

widely employed in this country; its chief use has been in connection with the treatment of hypertension in England and France. A patient, for example, with a blood pressure of 206/110 has an average pressure of 158; after a given course of treatment, his pressure changes to 188/100 with an average pressure of 144. There has thus been a drop of 14. It was considered that a single number was more informative and simpler to use than stating both levels. However, the single number index gives no data concerning which level (systolic or diastolic) has changed the more, a finding which has some practical significance Average blood pressure has also been used in certain types of anesthesia and in space medicine research.

Mean blood pressure must be distinguished from average blood pressure just discussed; mean blood pressure is a physiologic concept concerned with the pressure which the arteries would sustain if blood flow *were steady* and not pulsating. Arterial pressure is near the *systolic level* for only a relatively short period during the cardiac cycle; during most of the cycle it is near to the *diastolic level*. Mean arterial pressure is thus *closer* to the diastolic level than systolic. Determination of absolute *mean* pressure requires complicated electronic equipment but relative mean pressure can be estimated by the Reache formula which states that pulse pressure divided by 3 added to diastolic pressure will approximate actual mean pressure within a plus-minus error of 5%.

The simple formula becomes:

$$\frac{\text{SP (systolic pressure)} - \text{DP (diastolic pressure)}}{3} + \text{DP} = \text{Mean Pressure}$$

Thus a subject with a blood pressure of 134/80 has a mean pressure of 93:

$$\frac{SP(134) - DP(80)}{3} + DP(80) = 93$$

Mean blood pressure emphasizes the importance of diastolic pressure in various types of hypertension. For example, in aortic insufficiency with a blood pressure of 240/20 mean pressure is only 93 and within normal physiologic limits; whereas another subject with a pressure of 240/120 has a mean pressure of 160 which is the danger zone. The practical importance in determining mean blood pressure is in relation to prognosis and treatment in hypertension; the higher the mean pressure, the more serious is the outlook. Thus, the patient with a pressure of 240/20 with a mean pressure of 93 has a much brighter prognosis than another man with a pressure of 180/110 and a mean pressure of 143. The time factor and pulse rate in mean pressure has not been considered in the Reache formula; when the pulse rate is slow as, for example, 60 per minute, there will be only half as many high systolic points than when the rate is at 120. Although the systolic point is very brief, doubling the number of impacts in the vascular tree is more likely to cause additional wear and tear than the slower rate. A recent modification of the Reache formula adds a factor (about 20% of the pulse rate) in recognition of the time intervals of systolic impact. Thus, the papatient above with a pressure of 180/110 and a mean pressure of 143 will have a Timed Mean Pressure of 157 with a rate of 70, 163 at 100, and 167 at a rate of 120. It is probable that both mean blood pressure and timed mean

blood pressure will assume greater clinical significance with the development of new drugs for the treatment of specific types of hypertension; as a clue to prognosis both have suggestive implications in the physical examination of the cardiovascular patient.

A word should be said concerning blood pressure determination in the legs; when the pressure cuff is applied to the upper leg, arterial sounds may be heard with the stethoscope over the popliteal space at a point just below the head of the tibia. No problems are presented in thin subjects but heavy individuals with thick thighs are poor candidates for femoral blood pressure examination. Most arm cuffs are too small to encompass the area and even the long tailed type may be impossible to adjust properly. Femoral systolic blood pressure is normally about 20 to 30% higher than brachial pressure while diastolic pressure remains about the same. The indications for femoral blood pressure determination are mostly related to the peripheral vascular disturbances. Intermittent claudication, for example, may be due either to a diminished local arterial blood supply or to a general reduction of blood to the entire leg; the obstruction may be in the tibials, femorals, or high in the bifurcation. A subject with a femoral blood pressure of 180/88 on one side and 140/86 on the other, has a much greater problem than when both pressures are within the normal range of 180/88 and the obstruction is lower down. In the shock phenomena, frequently associated with acute myocardial infarction, it may be difficult or impossible to determine brachial blood pressure. In selected patients, femoral blood pressure has some significance in diabetes; in cases with less

than normal relationships with brachial pressure, the hazards of peripheral vascular disease with possible gangrene are enhanced. The drop in the femoral pressures may occur many months or years before symptoms of peripheral vascular disturbance are manifest. If possible, femoral blood pressure determinations should be performed on every diabetic patient; the time spent may yield future benefits.

CHAPTER IV

X-RAY EXAMINATION OF THE HEART

Visualization of the heart and aorta is probably one of the most rewarding experiences of the entire cardiovascular examination. Nearly all the other methods of approach are based upon deductive or inferential data; even a fleeting glimpse of the living pulsating heart may present more factual information than a score of tangential tests. In the old X-ray department at Boston City Hospital in the year 1910, Dr. Francis H. Williams placed a plaque on the wall with the simple motto. "SEEING IS BELIEVING"; in practical cardiology, nothing can equal the actual seeing of the heart in the performance of its work. Fortunately, X-ray equipment has become available to most practitioners; the minimum apparatus is a fluoroscope which is within the financial range of other office equipment.

Since the heart is a dynamic organ with changes in size and contour taking place in fractions of a second, fluoroscopy becomes more important clinically than roentgenograms. Besser compared the relationship to a single frame in a motion picture film; the appearance of the heart on any film represents but a tiny part of its cardiac activity captured perhaps in one hundredth of a second. Kymo-

graphic films are recognition of the limitations set by a single view of the cardiac cycle.

The heart and aorta readily lend themselves to X-ray study; surrounded by the highly radiolucent lung tissue no other organ of the body enjoys such normal contrast media. It is possible to examine in great detail most of the contours as well as the pulsations of various chambers of the heart and the movements of the great vessels.

Exact measurements of the heart and aorta are possible by both orthodiagraphic fluoroscopic methods and teleoroentgenograms; since X-ray follows the general laws of light transmission, the size of a shadow cast by an object will depend upon its distance from the light source and the receiving surface upon which the shadow falls. When the distance between the object and the screen is constant the shadow becomes larger as it approaches the source of light; object and shadow are the same size at a given distance which can be calculated from a formula. In X-ray determination of heart size this distance is 2 meters or about 6 feet; the standard teleoroentgenogram is a 6 foot film.

The orthodiagraphic method substitutes a central "pencil" of X-rays instead of distance, in order to obtain a parallel source of propagated waves; when a source of parallel X-rays is moved around an object, the shadow which is cast will be exactly the same size as the object itself. In ordinary fluoroscopy, the X-ray tube is focussed upon the center of the screen; both tube and screen move together in a fixed position. In orthodiagraphy, the tube carriage and screen are hung separately so that both have independent freedom of motion. In practice, the screen

is held fixed to the subject's chest and only the tube moves. The margin of the heart and aorta are traced directly upon the screen with a suitable marking pencil and later transferred to a special type of tracing paper; the finished product is known as an orthodiagraphic X-ray tracing. Special attachments may be added to the fluoroscopic apparatus by which a tracing is made with a pantographic mechanism; such equipment is rather expensive in comparison to the simple change in the separate hanging arrangement of the X-ray tube and screen. The conversion can be made by the average skilled craftsman; many practitioners have made the change themselves.

Both methods of determining exact heart size present problems. Six foot films, because of the great distances involved, lose much of the detail which may be important; the edges of the cardiac and aortic shadows tend to be fuzzy and indistinct so that measurements may have errors in the range of 2 to 5 mm. X-ray films must be processed, even under the most optimum conditions there will be a time lag of 20 to 30 minutes or longer between the exposure and the opportunity to examine the film. It is difficult to make the necessary measurements on a wet film, even the quick drying process requires an additional 20 to 30 minutes in some cases. Teleoroentgenograms present filing problems; they cannot be folded and kept with the patient's other records. The film and its processing represents certain fixed charges which must include the dark room budget and technician's time. Against these disadvantages is the one positive fact that 6 foot films are an objective and purely realistic presentation of the heart and aorta completely uninfluenced by the personal equa-

tion which may play a great part in orthodiagraphy. Moreover, far less experience and training are necessary to produce satisfactory teleoroentgenograms.

On the other hand, orthodiagrams provide on-the-spot measurements and diagnosis; the complete tracing can be made within 5 minutes. The procedure is relatively inexpensive; the tracing can be folded and placed in the patient's records. There are no dark room or filing problems. The method lends itself to the functional studies which will later be described. No time is lost in waiting for data and the examination can be repeated without further preparation. The chief disadvantage and perhaps the only valid one has been hinted already; this is in relation to the experience and skill of the examiner. Unfortunately, orthodiagraphy is not understood and learned overnight and the method demands more than the knowledge required to push buttons and turn switches. A long period of training followed by some years of experience may be necessary to produce a satisfactory orthodiagram. The personal equation and the unavoidable tendency to under- and overemphasize certain features of the examination to conform with pre-existing ideas is always an integral part of every orthodiagram; it can never approach a six foot film in objectivity and factual presentation.

Regardless of the method used for X-ray mensuration of the heart and aorta, preliminary fluoroscopic examination must be made before any attempt to estimate the various diameters is considered. It need not to be emphasized here that in using any source of radiation equipment, the necessary and approved protection measures are mandatory for the patient, doctor, and others who may be

present in the examining room. Maximal exposure limits have been determined by several agencies interested in the various types of radiation hazards in medicine and industry; these standards should be known and respected and not less than annual tests are advised concerning the possibility of accidental radioactive effects.

Preparation of the patient as well as the examiner should follow a carefully thought out routine which must follow a step by step plan which every practitioner will develop over the course of time. For the patient, a minimum of clothing is required; full exposure of the chest is mandatory. Even the slight shadow cast by a patient's gown may be confusing in certain cases. The subject should be placed at ease with a brief explanation of the procedure; dark rooms, eerie lights and sounds, an atmosphere of scientific anticipation, do not contribute to the average patient's peace of mind. The paroxysmal disturbances have been known to occur under such periods of psychosomatic stress. One of the most distressing incidents that may occur during fluoroscopy is syncope and collapse of the patient; such an experience in the dark room is not likely to be forgotten by either doctor or patient. If contrast media are employed during the examination, the patient should be shown the barium or other mixture before the lights go out and a short rehearsal of what is expected may expedite the period of examination.

Preparation of the examiner begins with adequate dark adaptation; the red glasses or goggles should be in place at least ten minutes. Proper adaptation reduces the need for higher radiation intensity. Total exposure time should be noted at the end of the examination. A recent statement

from the Atomic Energy Commission suggests that this exposure time should be noted on the patient's record; it may be mandatory by 1960.

When all is in readiness, the patient is first examined in the frontal or P-A plane with the subject facing the examiner and hands on the hips; the position of the patient's hands is important. The first rule in the dark room is to keep these hands out of mischief; panicky individuals may grasp anything in the dark, including the X-ray control panel or anything within reach. Even with shockproof equipment, unpleasant and perhaps dangerous situations could develop. Hands on the hips permit the patients to perform the deep and forcible respiratory tests, which are a part of the examination, in the most efficient position. It may be wise, in certain cases where the subject is perhaps slow in comprehending what is required of him or in apprehensive persons, to practice the breathing exercises a few times in the lighted room. If there is a language barrier so that the patient does not thoroughly understand the circumstances, time must be taken out to clarify the situation. If some of these details appear to be unnecessary and repetitious, it may be pointed out that even the most carefully prepared projects in science may be ruined by an otherwise insignificant mishap; bitter experience has shown that it is usually the small rather than the large errors which invalidate any given technical procedure.

Fluoroscopy in the P-A position is initiated by a rapid survey of the entire thoracic cavity; examination of rib cage and spine for asymmetrical changes is of first importance. Scoliosis and deformities of the spine are common

in the middle and older age groups; these may not be evident on previous inspection. Moderate and advanced orthopedic conditions of the spinal column may cause a shift in the mediastinum with change in position of the heart and rotation of the arch of the aorta. A predominate convex scoliosis toward the right may cause a retraction of the heart toward the right and backward while a similar convexity toward the left produces a shift to the left and forward. This observation has correlation importance with axis deviation in the electrocardiogram when the electric axis of the heart follows the plane of the anatomic axis. A shift to the right and posterior may change a normal axis pattern into a right axis and a structural left axis may be neutralized. The reverse is almost true for the shift toward the left and anterior; a normal axis becomes left and a previous left becomes more pronounced but a previous right may remain more or less unchanged.

The lung fields are now studied for unusual markings. Increased density and markings of the hilar zones may occur early in changes of pulmonary pressure; it is seen in mitral stenosis when left atrial pressure gradients become greater than ambient pulmonary venous pressure. This may develop rather rapidly in certain types of rheumatic infection of the valve structure and it remains as a rather constant finding throughout the life of the patient; it may be a residual pattern even after commissurotomy. Studies made after surgery usually show a marked drop in wedge pressures obtained from the pulmonary artery but the increase and accentuated hilar markings do not ordinarily disappear. Increase in hilar density and markings is also found in mitral insufficiency but this may be a

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ant in relation to vital capacity and other function tests; bnormalities should receive attention. On deep expiraion, the movement of the diaphragm upwards should lso be carefully observed; there may be an unequal rate f this movement. Unequal speed is usually more noticeible on expiration than inspiration and is more common on the right than the left. Such differences occur in certain ung diseases but it may be seen in congestive failure.

Changes in position and contour of the heart may occur during the deep respiratory tests; this is especially noted in subjects with the high diaphragm syndrome. In this condition, a gas distended transverse colon particularly in the splenic flexure pushes the left diaphragm and entire mediastinum upwards. The heart is shifted toward the left and rotated posteriorly; both the anatomic and electric axis may thus swing to the left. This produces the so-called functional or positional type of left axis deviation in the electrocardiogram. On deep inspiration, a heart which appears widened will tend to drop further than the diaphragmatic excursion; in straightening itself out, a previous wide shadow may return to a more normal contour; with this change in position, axis deviation may be reduced or entirely disappear. In the section on practical electrocardiography, the deep inspiratory test is suggested in all cases where a left axis deviation is noted at the time the third lead is taken. Misinterpretation of the axis pattern may be lessened by this simple procedure. Changes in size and shape of the heart are more common in the wide thorax type than the narrow during the deep inspiratory test. This will be considered later in relation to the Thoracic-Pelvic Index of Wolffe. Observation of

late discovery in comparison with mitral stenosis. Such hilar changes are confirmatory evidence of organic disease in debatable cases presenting a systolic murmur in which functional characteristics have been noted; functional murmurs rarely produce such changes.

The right pulmonic artery should be visualized in every patient; *the physician should familiarize himself with its* position from a study of angiocardiographic X-ray films of the normal subject. It has a fairly constant relationship with the junction of the right contour of the heart with the beginning of the aorta. It presents an important measurement in the 6 foot film or orthodiagraphic tracing; it normally is about 14 mm. or less in width. An increase *suggests an elevation of pulmonic pressure and it is* a more or less constant finding in cor pulmonale of both the cardiac and pulmonic types.

Increased density of the entire lung field is seen in chronic diffuse pulmonary stasis secondary to congestive failure; the density is usually more marked at the bases where it must be distinguished from collections of fluid. *Pulmonary and pleural disease should be noted;* areas of emphysema tend to brighten the lung fields while all other pathologic conditions present increased densities.

The respiratory tests should now be performed; the subject is instructed to take a deep breath and hold it. Movements of the diaphragm should be noted; the fall on both sides should be equal but the right may be greater in heart disease. Pleural and pericardial adhesions may restrict free movement of the diaphragm; the restriction may be in local areas or involve an entire leaf. Movements of the ribs and changes in the intercostal spaces are impor-

the entire border. The lower border of the heart is important in mensuration; in certain cases it may be necessary to increase the gas content. As described later, this may be easily accomplished by the drinking of a carbonated liquid; ginger ale or club soda water can be used.

Enlargement of the various chambers of the heart can be noted in the P-A position. On the right border, in cases of advanced mitral stenosis, the left atrium may appear at the point where the two major curves meet; it may be recalled that the traditional use of the words "right" and "left" were originally employed by the sixteenth century anatomists in reference to the position of the ventricles *in situ*; the atria are actually anterior and posterior. When the heart is approached surgically, the right atrium is the presenting part as the pericardium is opened; the left atrium is almost entirely posterior. When it hypertrophies or dilates, the enlargement may be seen both on the right contour and the left. On the left, it appears between the pulmonic curve and that made by the left ventricle. The contours produced by the left atrium on both sides may further be identified by the timing of the pulsation, since this occurs in late diastole or presystole, the movement will be inward just before the ventricles start their contraction. The largest movement and the greatest excursions occur at the apex; the inward movement of systole is quicker and more vigorous than the outward movement of diastole. The normal cardiac movements should be observed until the pattern is well remembered; change in the pulsations of the various chambers and great vessels may be demonstrable before any other clinical evidence of a specific pathology is noted. Of special interest in this

the changes produced during respiration as well as those occurring during the cardiac cycle will emphasize the previous statement that an X-ray film of the heart may well be likened to a single frame in a motion picture sequence.

With completion of these preliminary observations, the heart and aorta are now ready for examination. In the frontal or postero-anterior position, certain landmarks should first be identified. The significant ones on the right contour are two curves; the lower one arising at the diaphragm is a shadow of the right atrium; the upper curve is that of the ascending aorta. The indentation where the two meet is a point of some importance in mensuration. On deep inspiration, a small triangular shadow may appear at the junction of the diaphragm at the right atrium; this is the inferior vena cava. The superior vena cava is frequently seen as a shadow running parallel and to the right of the aorta. On the left there are three significant curves; the upper one is produced by the knob of the aortic arch as it turns posteriorly and to the left, the middle one which is the least prominent is the outline of the pulmonic artery, and the lower which is the largest is the lateral border of the left ventricle.

Pulsations should be noted; inward movement of the left ventricular border and an outward movement of the aortic and pulmonic artery curves take place in *systole*. Where the two movements meet is known as the point of adjacent opposite pulsation. About one-half of the lower border of the heart is obscured by subdiaphragmatic structures; on deep inspiration, an additional margin may be disclosed. A gas filled stomach or colon may reveal nearly

heart disease, with congestive failure, dyspnea, and unsteadiness, in large and obese subjects, and in women with massive breasts, the simple turning into a given position may present a number of complications. The situation may also be aggravated by difficulties encountered in raising the arms for certain routine positions. Experience has shown that, unless the patient is given as much assistance as possible and unless a minimum of energy is required, the examination will not yield enough information to make the task worth the effort expended both by the patient and the examiner. It should be repeated that weakened individuals standing in an awkward position surrounded by "dangerous" apparatus in a dark room, require the utmost encouragement concerning their physical and mental comfort; to this end some type of support is necessary. The first is related to appropriate devices to which the patient may grasp while being examined. Manufacturers have been slow in recognizing the need for such handles or rails but the physician may easily install them; parallel handrails may be placed along the edges of the fluoroscopic apparatus. A rotating platform about 18" in diameter makes a convenient turntable upon which the subject can stand and be turned at the will of the examiner; in mensuration where accuracy of position is mandatory, a suitable gearing device will permit positioning at any given angle without movement of the patient. A number of these are available and may be purchased but the physician can make his own equipment as a matter of expediency.

Rotation of the patient will reveal a changing series of cardiac contours; certain profiles of the heart and great

connection is the fluoroscopic discovery of aneurysmal dilatation of the left ventricular wall as a late manifestation of myocardial infarction. This will be discussed later but it may be pointed out here that this syndrome can be noted early in its development by observation of an area of reverse movement of the left ventricular border just above the apex; with the excursion of the contour inward a more or less small area appears to bulge outward. This type of paradoxical pulsation occurs whenever a *thinned out area of the heart muscle is unable to* withstand the strain of systolic pressure; replacement of infarcted heart muscle by scar tissue is a recovery phenomenon in every surviving case of coronary occlusion. Aneurysms are most likely to occur where the pressure factors are greatest and they are best seen along the left borders of the heart, sometimes as a solitary finding with no ECG changes or other diagnostic abnormalities.

Other Positions

While the frontal or P-A is the most important position in X-ray examination of the heart, significant clinical data may also be obtained by observing the cardiac shadow from different angles. The subject can be turned throughout a complete circle and many of the various contours of both the cardiac chambers and the great vessels may be studied *in detail. Turning the patient may not always* be a simple procedure; the normal or nearly normal individual standing in front of the fluoroscope ordinarily presents no problems. He takes the position himself after instruction from the examiner. In patients debilitated by

MEASUREMENT OF THE HEART AND AORTA

Estimation of the size of the heart, its various chambers, and the aorta has been made possible by the development of a number of simple basic formulae. Correlation between such studies and post mortem material has shown an accuracy of a plus-minus error of about 5% in the hands of competent examiners. Absolute size is of far less clinical importance than relative size; the relation of the heart size to a given subject has more clinical implications than a relationship to other members of the comparative group. Cardiac mensuration receives its greatest importance in relation to changes which take place in the patient himself; whether his heart is larger or smaller than that of another man of equal age, weight, height, body build or blood pressure is of academic interest compared to the determination of changes which occur over a period of time in his own heart size.

Cardiac mensuration has suffered the burden of two points of view, those who are primarily interested in insurance, industrial, compensation and military medicine, seek information concerning the size of the heart and aorta in relation to a given "normal" for the group. A heart which is larger than this so-called "normal," immediately places the candidate in a suspect class and he may be rejected on this finding alone. The difficulty in establishing such "normals" has led to some questioning concerning the validity and importance of cardiac mensuration; the number of variables has made the task al-

vessels have been more or less standardized as, for example, those seen in the left and right anterior positions. But other views may have equal importance; the lateral right-left position permits measurement of the lateral or P-A diameter of the heart and the barium filled esophagus is occasionally better viewed in a half right anterior position. The position and the characteristic pulsation patterns of the various chambers and great vessels in health and disease require a period of study and observation which will, of course, vary with each examiner; fortunately a number of visual and other aids which have been developed over the past 30 years make this task less formidable.

Enlargement of each of the various chambers of the heart *and the probable pathology responsible for* the condition may be determined by fluoroscopy; changes in the aorta, pulmonic artery, and the venae cavae can also be discovered. The profile of congenital heart disease with the specific lesions present can also be noted. Pericarditis, mediastinal changes, and upper thoracic involvement may be studied. The barium filled esophagus permits establishment of important relationships in certain types of heart disease; in mitral stenosis, for example, the increased size and shape of the left atrium may be clearly visualized, while in aneurysmal changes of the aortic arch, the enlargement may be estimated. Description of these findings are the subject matter of large textbooks and no attempt is made here to consider any syndrome in the detail required for a complete resumé of the wealth of clinical X-ray material *which has been collected in this* special field of medicine.

below; the ordinary X-ray illuminating box fixed in a horizontal plane can be used. If tracings alone are employed, a flat desk or table with a glass or transparent plastic top under which a simple measuring grid may be inserted, will expedite measurements.

The Chief Cardiac and Aortic Diameters

The diameters of the heart and aorta are first drawn on the tracing; for teaching purposes and for objective clarity two or three colored pencils are useful but not entirely essential. The mid-thoracic line is drawn from the mid-clavicular space to the xyphoid point; the largest diameters of the aorta and heart from this midline are then drawn in with a red pencil. The widest diameter of the chest is also drawn to the midline in red In the author's clinics, these are known as the "red diameters" and are related to the thoracic ratios. With a blue pencil the contour of the heart is completed at the upper and lower margins, since the heart is a symmetrical organ, these margins will approximate the actual borders in most cases. The lower border can almost be completely visualized by the gas filled stomach procedure The purpose of outlining the cardiac oval is for the later determination of the frontal area of the heart and for the three dimensional calculation of size; the longest diameter of the oval and the shortest are drawn in blue. The third or P-A diameter which was observed on lateral X-ray examination is also marked in blue. It is customary but not necessary to place this diameter below the surface area. It could be indicated

most impossible since many normal persons in a similar group exhibit wide differences in heart size. In borderline cases no competent opinion can be rendered. The opposite point of view is held by those who are interested in the individual as a cardiac patient; here the determination of heart size may spell the progress of the disease and the effect of treatment. The large dilated heart, in congestive failure for example, which gradually becomes smaller as a result of diuresis and digitalis therapy can be used as an objective index of prognosis. This conflict of interest has in the past been an unfortunate hindrance to the full employment of a valuable clinical procedure. The author recognizes both points of view; he has had experience in attempting to formulate standards from the X-ray examination of many thousands of normal Navy and Marine personnel, he has been unable to develop a satisfactory formula to distinguish the median variant between normal and abnormal heart size.

It has been pointed out that the importance of heart size in any given subject is in direct relationship to his other body measurements. The size of the thorax, for example, is an important relationship as well as the size of the body itself. The size of the thorax and pelvis may determine the configuration and electric axis of the heart. There are other correlation factors which may be determined by simple formulae and all together a comprehensive survey of the heart and aorta is made possible.

Actual measurements are made upon the six foot film or the orthodiagraphic tracing. In practice, the film or tracing is placed upon a flat surface illuminated from

below; the ordinary X-ray illuminating box fixed in a horizontal plane can be used. If tracings alone are employed, a flat desk or table with a glass or transparent plastic top under which a simple measuring grid may be inserted, will expedite measurements.

The Chief Cardiac and Aortic Diameters

The diameters of the heart and aorta are first drawn on the tracing; for teaching purposes and for objective clarity two or three colored pencils are useful but not entirely essential. The mid-thoracic line is drawn from the mid-clavicular space to the xyphoid point; the largest diameters of the aorta and heart from this midline are then drawn in with a red pencil. The widest diameter of the chest is also drawn to the midline in red. In the author's clinics, these are known as the "red diameters" and are related to the thoracic ratios. With a blue pencil the contour of the heart is completed at the upper and lower margins, since the heart is a symmetrical organ, these margins will approximate the actual borders in most cases. The lower border can almost be completely visualized by the gas filled stomach procedure. The purpose of outlining the cardiac oval is for the later determination of the frontal area of the heart and for the three dimensional calculation of size; the longest diameter of the oval and the shortest are drawn in blue. The third or P-A diameter which was observed on lateral X-ray examination is also marked in blue. It is customary but not necessary to place this diameter below the surface area. It could be indicated

elsewhere on the tracing. These "blue diameters" are used in the calculation of surface area, total heart size, and in the cardio-body index. Finally, the root of the aorta previously observed in the right anterior position and placed for convenience near the left outer clavicle, is marked in green and a similar diameter is made across the right pulmonic artery.

Addition of the right and left horizontal diameters of the heart will give the total width of the heart or the transverse cardiac diameter. In a similar way, the addition of the right and left aortic diameters will give the total width of the aorta. With these figures it is now possible to determine several relationships concerning relative heart size.

The transverse diameter of the heart is calculated by the formula:

$$TR + TL = \text{Transverse Diameter}$$

where $TR = $ right projection from midline (red)
$TL = $ left projection from midline (red)

Tables of average normals based upon height and weight have been developed for orthodiagrams by Hodges and Eyster and for teleoroentgenograms by Ungerleider and Clark; these tables while showing errors in certain height groups, are the best available for examination of the average American. *Since these studies were made more* than 20 years ago, there has been a change in the size of the native born subject who is generally larger than his parents.

The width of the aortic arch is determined by the formula:

$$AR + AL = \text{Aortic Arch Diameter}$$

where AR = maximum extension to right border of arch from midline (red) and AL = maximum extension of left border from midline (red).

The same tables can also be used for estimation of relative width of the arch; normal variations occur within a minus 5% and a plus 12% of the given measurements. Corrections are made for age.

Standards based on length and breadth have also been determined for the surface area of the heart. Here the formula is:

$$A = \pi/4 \, L \times B$$

where L = the long diameter of the heart (blue)
and B = the broad diameter of the heart

Actual and more accurate measurements of the surface area can be determined by the Free-Hyman planimeter which is a modification of the ordinary instrument employed by engineers and architects and adapted for special use in cardiac mensuration. The planimeter is adjusted to a given point on the orthodiagram usually at the junction of the diaphragmatic angle and the right ventricle on the right while the moving leg of the instrument traces the cardiac oval. The area is read directly in square centimeters from the dial. Many practitioners prefer direct measurements with a planimeter than the employment of the calculated tables; the instrument is not expensive, is simple to use, and will last a life time with ordinary care.

In addition to the absolute figures obtained in regard

to the aortic and cardiac diameters and the surface area of the heart, a number of relative index figures or scores may be secured by comparison of certain factual data. The indices, as indicated before, may have more clinical value insofar a given patient is concerned than his cardiac relationship with the average individual in his particular group. Moreover, since each subject presents his own standard, deviations are readily discovered. The most useful and practical of these index values will now be considered in some detail.

Cardio—Thoracic Index

The relationship between the width of the heart and the width of the thorax is the oldest cardiac measurement; it is the simplest but also the least informative. When the width of thorax is divided by the width of the heart (transverse diameter) the average index is about 55% with normal variations extending from 45 to 70%. The index has been largely abandoned because of this wide band of indeterminate normals and it has little value in comparing a given subject with this group. The Cardio-Thoracic Index, however, retains considerable importance in relation to the patient himself; for example, if a subject has a transverse diameter of 11.4 cm. and a chest width of 23.6 cm., the index will be 47%. This is well within accepted normal limits; about a year later, after having experienced a bout of acute rheumatic fever, the transverse diameter is found to be 13.4 cm. With the thorax unchanged, the index is now 57%, an increase of 10%, but still within the upper normal limits. Assuming the thoracic diameter to

remain constant over relatively long periods of time (up to 10 years), the index will vary directly in proportion to the changes in the width of the heart and while this change can be noted in other tests, its simple application may have some importance in the cardiac examination.

Total Volume or Three-Dimensional Size of the Heart

The surface area calculations of the heart are based upon the frontal or P-A positional examination of the cardiac shadow. As the name implies, it is the estimation of heart size in only two planes, since the heart is a solid object with thickness as well as length and breadth, a third diameter is required to calculate its actual size or total volume. A number of formulae have been devised for this calculation but the one with the most practical value and simplicity of use is the Besser Index in which width multiplied by length multiplied by thickness and again multiplied by a factor will give the volume in cc. The factor used by Besser in 1940 was 0.455; subsequent corrections show the factor to be 0 498 but relatively little error occurs when 0 5 is employed. The revised formula thus becomes:

L (long diameter) × B (broad diameter) × D_3 × 0.5 = Heart Volume

D_3 or the third diameter is the transverse anteroposterior diameter measured in the right lateral position.

In actual practice, the three diameters of the heart are multiplied together and divided by 2. Total cardiac volume is the most accurate X-ray measurement of the heart; this can be proven experimentally by the simple water

displacement experiment. A favorite demonstration for students is the measurement of an unopened heart obtained at autopsy; the heart is then immersed in a full beaker of water with only the aorta and pulmonic artery exposed above. The heart is removed and the amount of water displaced is measured; the calculated volume of the heart and the calculated water displaced usually agree within 2 to 5%.

Total volume of the heart thus becomes an important method of determining small changes in cardiac size; just as surface area is more accurate than the cardio-thoracic index, so 3-dimensional determinations are more accurate than 2-dimensional. This is particularly true when the enlargement is chiefly in the lateral anteroposterior diameter; there may be considerable enlargement of the heart with relatively minor changes in the surface area measurements. Thus, a girl of 18 with acute rheumatic fever showed a long diameter of 13.7 cm., broad diameter 102, and lateral diameter 9.6; surface area was 108 cm. and *the total volume was 720 cc. About one month later the* long diameter was 13.6, broad 10.8, lateral 11.4; the surface area had increased to 115 cm^2, which represented a 6% enlargement while the total volume increased to 845 cc. or an overall enlargement of 17%.

Cardio-Body Index

The relation of the size of the heart compared to the size of the body has long had academic interest; this relationship has significant physiologic implications. It would appear obvious that, everything else remaining constant,

a larger body would require a larger heart; in nature, larger animals have larger hearts than smaller animals although the relationship by weight or size is not a symmetrical curve. Studies of normal subjects show a wider variation when the surface area of the body is compared with the surface area of the total heart than when body surface area is compared with the frontal surface area of the heart. This has led to the adoption of a formula which is not scientifically correct since unequal factors are employed but which has many clinical applications. The determination of a total surface area of the heart *in vivo* presents many difficulties; although the heart approaches a cone in shape, the many variabilities which are present have not permitted the development of a satisfactory formula from available data. In experimental work, the range of error in normal hearts is greater than some of the changes occurring as the result of disease processes. The comparison of surface area of the heart and surface area of the body, on the other hand, is a simple procedure; thus the formula becomes:

$$\frac{\text{Surface area of heart}}{\text{Surface area of body}} = \text{Cardio-Body Index}$$

The normal ratio extends from 450 to 550; it tends to be less in women and more in men. Surface area determinations of the body are obtained from the standard Dubois charts used in basal metabolism determinations and for other function tests.

The Cardio-Body Index is of value in the discovery of hypoplastic hearts of the type seen in the syndrome formerly called neurocirculatory asthenia but now classified as

psychosomatic cardiopathy; the small asthenic heart associated with certain diseases like tuberculosis, chronic wasting conditions, and certain psychiatric syndromes, may reach an index as low as 375. High normal Cardio-Body Index levels are seen in well conditioned athletes; here the index may rise to 600 in the smaller body types.

Pathology is nearly always present when the index rises above 625; high levels like 900 to 1400 are seen in congestive failure due to valvular heart disease and in hypertension. In Chapter VIII, a patient with an index of 1650 is shown in relation to the X-ray exercise tolerance test. In summary, the Cardio-Body Index is a method of determining heart size in relation to body size; its current employment is perhaps more confined to research work than clinical medicine. It is now playing an important role in space medicine but there are many applications of its use in practical cardiology; it provides an individual yardstick in the treatment of chronic cardiac disabilities like those associated with rheumatic infection, hypertension, and post coronary syndromes.

Aortic Index

The determination of total width of the aortic arch and its comparison with standard tables have already been presented. Further data can be supplied from examination of the root of the aorta as measured in the right anterior position. The total width of the aorta represents the combined width of both the ascending and descending portions of the arch; in normal subjects examined at autopsy both portions have about same width. If one por-

tion can be easily visualized by X-ray, a relationship can thus be established which may have diagnostic implications. In general, the root or ascending portion is more readily measured than the descending; when this is compared to the total width an Aortic Index can be established. Normally, the index is 50% since both portions are the same but increase in the width of the root will cause a rise in the index level. Thus, if the total width is 6.1 cm. and the root 4.3 cm., the index will be 70.5%; if the descending portion is wider, the index will fall to 44.4% if the total width is 6.1 cm. and the root is 2.7 cm.

Stated from the point of view of specific aortic change, if the aortic index is greater than 50% the root and ascending portion is probably enlarged while, if the index is less than 50%, the descending portion is bearing the brunt of the pathology. Experience has shown that increase in size of the root and ascending aorta is likely to be due to luetic disease while changes in the descending portion are most often caused by atheromatous and arteriosclerotic pathology. The aortic index may thus be useful in differential diagnosis of various types of aortitis; an increased index suggests syphilitic involvement while a lowered index is usually associated with the degenerative diseases of the aorta. The greater the deviation from the norm of 50%, the more accurate is the diagnosis likely to be. Aneurysmal dilatation of the root and ascending portions of the aorta may show an index as low as 15%; aneurysmal changes of the descending portion may raise the index as high as 85%. Where both limbs of the arch are involved, as in advanced stages of both types of pathology, the index will have little significance.

Aortic-Thoracic Index

The relationship of the total width of the aorta and the width of the chest is in the same category as the Cardio-Thoracic Index; it was developed about the same time and is subject to the same objections. The average normal was considered to be about 35%; the aortic arch is usually about one-third the size of the thoracic width. It is more widely used in Latin America and Europe than in this country. It has a proven practical value in estimating the progress of aneurysmal dilatations of the aorta; a rising Aortic-Thoracic Index from a previous 65% to 75% may carry a more sinister significance, for example, than the simple statement that the width has increased 0.6 cm. From this point of view the index should be remembered by the physician for use in selected cases.

Right Pulmonic Artery

It has been previously noted that the right pulmonic artery is readily visualized at or near the right border of the heart; angiographic studies have shown that this vessel is a short, sharply angulated artery which branches off the main pulmonary artery just posterior to the shadow cast by the root of the aorta. It is best seen as it crosses the fifth or sixth or sometimes the seventh rib posteriorly; the added density of the ribs lend a contrasting appearance with the lung tissue and it can be measured without difficulty. Experimental and routine angiographic investigation has shown a relationship between the width of the

artery and pulmonary pressure; increase in pressure is accompanied sooner or later by increase in width. The average width in normal subjects is 14 mm. or less; the figure 14 is rather constant in adults. In mitral stenosis, advanced types of mitral insufficiency, in cor pulmonale due to disease of the lung, and in few rare conditions with increased pulmonary pressure, the artery may widen to as much as 18 to 20 mm. There is a close correlation between pulmonary artery wedge pressure and increase in width of the right pulmonary artery. This relationship has many clinical implications in addition to its surgical importance; it may be used as an index of right ventricular strain before obvious enlargement of the right ventricle can be noted. It may occur before the development of right axis deviation in the ECG and it is a rather constant sign in backward congestive failure. As indicated before, the actual width of the artery does not diminish readily, even after surgery and after restoration of compensation by other therapeutic measures. It usually remains as telltale evidence of previous pulmonary hypertension and may be a valuable clue in subsequent diagnosis and treatment.

Thoracic Pelvic Index

While not primarily concerned with cardiac mensuration, the Thoracic-Pelvic Index has an important clinical relationship with the anatomic axis of the heart and axis deviation in the electrocardiogram. It is the chief correlation factor in the interpretation of axis deviation with body type in subjects with normal skeletal system; de-

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contents of the mediastinum and the heart will assume a more vertical position. The reverse occurs in the narrow pelvis, wide chest body type; here, the smaller pelvis does not permit a dropping down and the heart is held in a horizontal position by the higher left diaphragm.

In accordance with the formula:

$$\frac{\text{Width of Chest}}{\text{Width of Pelvis}} = \text{Thoracic} - \text{Pelvic Index}$$

when the chest is wider than the pelvis, the index will be greater than 1.000 as for example, in an athlete with a thoracic width of 36.4 cm. and a pelvic width of 28 5 has a T-P Index of 1.279. A woman with a chest width of 27.6 cm. and a pelvis of 32.4 cm. has an index of 0 854. In the normal subject, an index greater than 1.000 will have a tendency to be associated with a horizontal heart and hence a left axis deviation; the reverse occurs with an index less than 1.000 for here the tendency toward a vertical heart may produce a right axis deviation. The correlation curves are more consistent in the higher indices than in the lower; a heavy woman with a wide pelvis may have a normal left axis as the result of positional change due to causes leading to a high left diaphragm.

The Thoracic-Pelvic Index may be applied with clinical profit in any case where axis deviation is greater or lesser than anticipated from a consideration of other data obtained in the physical examination; it is especially important in determination of the clinical significance of left axis deviation in normal men.

formities of the chest and pelvis invalidate the index significance. *When the width of the chest is divided by the* width of the pelvic crests an index number is obtained. The width of the chest is obtained from previous X-ray determination; measurement of the pelvis may present some difficulties in heavy obese patients. The simplest method is to obtain the measurements at the time of the cardiac fluoroscopy; on the 6 foot film, the pelvic brim can be photographed at the top or bottom before or after the chest exposure, using the blocking out technique of Gottlieb and similar to that employed in spot X-rays of the gall bladder. In thin persons, the standard X-ray calliper may be used but it is impossible to estimate the subcutaneous fatty layer which covers the crests in the overweight individual.

Anthropologists recognize three body types insofar as the chest and pelvis are concerned; the first, is the large chest and small pelvis, the second shows a small chest and a wide pelvis, and the third is characterized by equal chest and pelvic measurements. A number of scientific names are given to these types but in general the wide chest, narrow pelvis type, is known as the male triangle with the apex down, while the narrow chest, wide pelvis, is the female triangular pattern with the apex up. The third type with equal chest and pelvis width is the neutral parallel type. *Since the bony structure of the pelvis is the* anatomic base upon which the viscera are chiefly supported, those subjects with a wider pelvis will have a tendency toward ptosis of the various organs of the abdomen and thorax. This downward pull will be reflected in the

be found between the various chambers. During the 1920's, the mathematical challenge offered by such measurements produced a wide variety of formulae which had a more or less definitive role in estimating capacity, volume output, relative pressure gradients, and functional defects of the valves. In their place and under special circumstances, nearly all have proved their clinical usefulness. With the advent of angiographic procedures, however, their employment has become increasingly less and few are in routine use. It is interesting, in concluding a final note on these older and perhaps forgotten methods of calculating chamber and other abnormalities from an orthodiagraphic tracing or teleoroentgenogram, that contrast media studies have subsequently verified many of the complicated deductions made by such pioneers as Besser, Williams, Groedel, and Holmes.

THE ANATOMIC AXIS

The long diameter of the heart (blue) used in determining surface area is usually drawn from the point where the right contour and the aortic curves meet at the apex. In normal subjects, this line represents with considerable accuracy both the anatomic axis of the heart as well as the electric axis as seen from a frontal plane. The angle made by this diameter as it crosses the midline of the thorax is the Basic Cardiac Angle; in normal individuals this angle is 47° (plus or minus 3°). The angle increases as the heart becomes more horizontal and decreases when it becomes more vertical. In enlargement of the left heart a correlation factor occurs between the developing left axis deviation and change in the anatomic axis; there is an increase in the cardiac angle. In marked hypertrophy, as in hypertension, the angle of the anatomic axis may rise to 60° or more. The correlation is less definitive in right axis deviation but small vertical hearts may show an angle of 38°.

The clinical importance of the angle of the anatomic axis is again in relation to electrocardiographic interpretation; in debatable cases it may add material information concerning the significance of certain otherwise unexplainable changes in the tracings. Determination of the angle is simple to perform; any type of protractor may be used.

OTHER MEASUREMENTS

Since the heart and great vessels lend themselves to accurate visualization, any number of relationships may

port, receives its importance only within the framework of clinical correlation; its interpretation is an electrocardiographic diagnosis, not a clinical diagnosis. In practice, a great gap may exist between the two; as indicated in Chapter VII, functional capacity of the cardiovascular system cannot be determined from an ECG tracing. Patients with grossly abnormal records may still be able to perform amazing feats of physical endurance while individuals with perfectly normal tracings may have a markedly reduced capacity for any effort. Patients have been known to die after passing through a long period of heart disease with congestive failure with no abnormalities in the ECG's and the reverse is equally common in patients who live many uneventful and unrestricted years with "ominous" changes in the tracings. The tendency to exaggerate the importance of electrocardiography will be discussed later but it may be said, here, that electrocardiography is but a single spoke in the great wheel of the cardiovascular examination; it may at times be an important spoke, but the wheel may still run well without it. A given tracing is therefore only as valuable as the physician's ability to utilize the specific information concerning the electrodynamic activity of the heart in a given patient in relation to all of the other objective data which he has obtained, assuming that the ECG record is free from technical errors, the experienced examiner does not question the physiologic mechanism responsible for abnormalities which may be present but he may well question the role, if any, which such changes play in the patient's cardiovascular status.

CHAPTER V

THE ELECTROCARDIOGRAPHIC EXAMINATION

We turn now to a consideration of the place of electrocardiography in the physical examination of the cardiac patient. It must be pointed out at the very beginning that this presentation makes no attempt to teach the fundamental concepts of this procedure. It will only be the intent of the author to discuss a number of practical problems which occur daily but which, unfortunately, do not receive the attention which they deserve and which may render useless or even hazardous, electrocardiograms.

The electrocardiogram is only as valuable as its interpretation; whereas any one trained in push button technique can take an electrocardiogram, a life time of experience may be required at times to venture an accurate diagnosis. Insofar as treatment and prognosis may depend upon adequate interpretation, the burden of clinical responsibility may lie heavily upon the practitioner in any given case. Interpretation rests equally upon the two important concepts; the first is the determination of the positive findings and the second, which has not been sufficiently emphasized, is the recognition of what the record *does not show.*

An electrocardiogram, like every other laboratory re-

trodes and the electrode connection; the electrode jellies and mixtures contain NaCl and other chemicals used to reduce surface resistance and fatty skin substances. These will produce corrosion areas on metal parts and unless wiped clean after use, faulty connections will result. Some of the fine tremors and irregularities in a tracing may be due to poor electric contact; such little tremors have been confused with the fibrillary movement seen in atrial fibrillation. Under certain conditions a corrosion contact will act like a condenser amplifier in the presence of 60 cycle alternating house current; all electrocardiographic apparatus are provided with a special grounding cable but this may not be sufficient to remove the amplifier effect of the so-called chemical sandwich which develops at the corroded electrode connection. The 60 cycle interference effect is a common occurrence in some direct writing machines. Parsonnet used to speak of the "spotless hygiene" necessary to maintain both electrodes and cable connections free from chemico-electric effects.

The possibility of misconnection of electrodes has plagued the physician from the very beginning of clinical electrocardiography. Errors in electrode connections have been called the calculated risk of every electrocardiographic examination, there are no "fool proof" methods which the manufacturers have been able to devise that can promise that an incorrect connection cannot be made. The simple expedient of marking the cables with the proper designation and the use of different colors assumes that the examiner will use judgment and common sense. Under the best regulated conditions and in the hands of able and experienced technicians, errors still

SELECTION OF THE ELECTROCARDIOGRAPH

Two types of electrocardiographic apparatus are available; the direct writing and the older photographic models. The direct writing instruments have freed the physician from the burdens of darkroom developing problems but they have also limited the scope of graphic study of the cardiovascular system. Nearly all of the photographic models have provisions for pulse wave recorders and heart sound devices; these are simple and valuable additions to the ECG which will be discussed later. To obtain the same information by the direct writing machines, additional channels are required with a considerable increase in cost. Moreover, photographic records are permanent, rugged, and present no filing or storage difficulties; direct writing machines use a delicate, friable, plastic foam covered paper which is easily scarred and which rubs off under moderate friction. The early types of such paper had a tendency to deteriorate under conditions of dampness and heat but the quality of the paper has continued to improve under the many research projects now in progress.

Like all fine precision instruments, the electrocardiograph demands gentle handling and constant care; the delicately adjusted mechanism is easily thrown out of alignment and strong handed manipulation may result in a badly damaged machine. Both the mechanical and electric systems require attention and as the apparatus is used over a period of time, parts will wear out. The most common technical difficulty is concerned with the skin elec-

looking through the back. Mixup of right and left arm connections in Lead 1, is perhaps the easiest to discover; reversal of the electrodes causes deflections of all waves downward which is an unusual pattern in adults. Other combinations require considerable study. The inadvertent use of the exploring electrode of the CF and V Leads in the limb lead hookup is rare; the employment of a misplaced limb electrode over the chest may cause a wide range of errors but sometimes these are difficult to detect as they may closely resemble the normal pattern. When such errors are suspected, the obvious procedure is to repeat the tracings under controlled circumstances. It is unfortunately true, however, that most electrode application errors are committed in hasty preparation of the patient; some irregularities change very quickly. For example, an individual with an episode of paroxysmal tachycardia should be examined immediately. In the attempt to get these records as soon as possible, there may be mixup in the electrodes; the error may be discovered too late and by the time the second examination is made the episode is over. The first tracing then becomes an interesting exercise in decoding.

Preparations of the Patient

Preparation of the patient is an important part of the examination; most poorly recorded tracings are due to failure of patient cooperation. Patient cooperation may be dependent upon many factors, many of which come under the direct responsibility of the examiner. Agitated, restless, frightened, and belligerent individuals are not

occur. The physician would be wise to adopt from the very beginning a step by step routine which will tend to keep these costly mistakes to a minimum. The following procedure has been effective for many years in some New York hospitals. The electrodes are always applied in a given order starting with the right arm, then the left arm, and finally the left leg. Cable connections are performed in the same order; when all connections have been made, an inspection is carried out in the reverse order starting with the left leg, working back to the left arm, and finally the right arm. Psychologic experiments made by Dr. A. A. Free, more than 25 years ago, showed that more misconnections were detected by reversing the inspection process than by following the original order since a repeated memory pattern had a greater tendency to overlook such an error. Where the apparatus is fixed in a stationary position, the chance of error is reduced to zero by the simple expedient of attaching the cables in position on the examining chair or table; the problem is thus chiefly confined to portable instruments.

Recognition of misplaced cables offers an interesting challenge in deductive reasoning. To the trained examiner, the tracing may present an unusual appearance which in many instances is difficult to define; the record simply "looks wrong." The old Winterberg mirror test is still of value in suspected cases; when the tracing is held upside down in front of a mirror, the reversed image is seen. If this image "looks more normal" than the original, there may be reasonable grounds to suspect an error in electrode connection. This same method can be used by holding the record upside down in front of a light with the observer

still retain a usefulness which cannot be supplemented by the suction cup electrode.

Ease of body is just as important as peace of mind in securing maximum patient cooperation. The position of the body may have great bearing in regard to the subject's comfort; a patient in congestive failure and orthopnea who requires 4 pillows or an elevated head rest in order to breathe with minimum effort, will not take kindly to a forced position demanding that he lie flat on a tabletop. Most electrocardiograms are taken in a recumbent position but they may also be obtained in a semi-reclining or sitting position. An all purpose reclining chair is available which permits examination of the individual in a horizontal plane and in all degrees to a sitting position; a simple substitute which has recently found wide favor, is the metal beach chair with a reclining mechanism. The physician, in his ingenuity may be able to rebuild or make a convenient table-chair combination which will accommodate the needs of nearly all subjects and which will establish satisfactory patient comfort.

The patient should be permitted to rest in the examining chair for at least 5 minutes before the electrocardiographic examination is made; the physical effort required to get to the examining room together with the emotional strain may be productive of various grades of tachycardia as well as the development of irregularities. The development of these findings is the purpose of the exercise tolerance tests but an original or primary tracing taken under basal conditions must be first obtained for comparative purposes. Where technicians have not observed these rules it is not uncommon to note a rate, for example, in

suitable subjects for the usual methods of obtaining ECG tracings; where such records are of vital importance special measures may have to be employed. Children may be "mummied" and adults may be confined in electrocardiographic straitjackets; this is ordinarily a hospital procedure but in the case of children it may even be necessary at the home or office. Such extreme measures may be justified in acute rheumatic carditis or chorea when a child cannot otherwise be managed; the task is worthwhile in establishing the degree and extent of cardiac involvement. Patients in coma and various grades of unconsciousness present no special problems unless involuntary muscle movements occur.

Most individuals approach their first electrocardiographic examination with some misgivings; the specter of accidental electrocution is always present and the application of the older strapped electrodes does nothing to lessen their apprehension. The newer suction cup applicators carry a less sinister significance to a nervous subject and their very simplicity recommends their routine use except in certain instances; when extensive varicosities cover the lower leg it may be unwise to apply suction cups. Ecchymoses and breakdown of the skin has occurred in some cases with subsequent ulcer formation; there has been one medico-legal action resulting from such a mishap. Patients with hypersensitive skin areas as in herpes zoster, periarthritis nodosa, and the arthralgias may object to the negative pressure effect of the suction cup. It may also be unwise to apply the cup in diabetic patients with suspected or evident skin infection or gangrene of the toes or feet. In all of these special instances, the strapped electrodes

each lead represents a particular pathway through the electrodynamic field developed by the living heart.

Which is the most significant lead? The question became an important one for manufacturers of operating room equipment who were attempting to supply the need for a cardiac monitor which would present to the anesthetist a constant record of the patient's heart action during surgery. The general consensus among cardiologists was that the first lead was more likely to show abnormalities when they occur than any other; oscilloscope cardiographs operating in lead 1 are now standard equipment for such work. If the physician for one reason or another is limited to a single lead, the lead 1 should be selected.

How many leads should be used for routine examination? In the examination of normal subjects as, for example, in insurance, pre-employment industrial work, and in sports medicine, where the number of individuals to be studied is very large, the conventional three leads and a precordial 4th lead has been considered adequate by the Cardiovascular Research Committee of the American College of Sports Medicine. In patients with heart disease or where a cardiac lesion is suspected, the 12 leads of the conventional and unipolar hookup are a basic minimum although recent studies have thrown some doubt concerning the need of the unipolar limb leads.

While the V leads have largely displaced the routine employment of the CF leads the older hookup may still yield information not discovered by the newer chest lead concept. Many cardiologists believe that the CF leads are more responsive in demonstrating lesions produced by myocardial infarctions located in the anterior surfaces of

lead 1 of 110 and a rate of 90 or less in lead V-6; likewise, irregularities seen in lead 1 may become less frequent or entirely disappear by the time the last lead is taken. It may be said that, as a general rule, if more changes in rate and rhythm are observed in the first lead compared to the last in any given electrocardiographic sequence, the *patient has probably not had a sufficiently long* resting period to produce a basal record.

How Many Electrocardiographic Leads?

The perennial problem facing the physician at every electrocardiographic examination is the question concerning the number of leads which should be taken; the choice extends from 1 to 31. The traditional answer has been that as many leads should be taken as may be necessary to either make a diagnosis or to exclude a suspected myocardial lesion. It has long been recognized that a positive diagnosis will require fewer leads than a negative one; all of the disturbances of conduction and irregularities of rhythm can be noted in one lead. A suspected lesion may not be discovered even when the entire combination of lead hookups is performed. The multiplicity of leads necessary in certain cases has been compared to the dissection problems presented to a pathologist attempting to locate a solitary lesion, for example, in the liver; a metastatic nodule might be discovered on the first cut through the tissue but it might require any number of sections before it was finally visualized. The electrocardiographic leads were originally described by Wenckebach *as electric cross sections* of the heart; the analogy still holds true, in that

routine examination of normal subjects may only need the 4 leads previously mentioned. A simple daily checkup of an arrhythmia like atrial fibrillation under treatment with digitalis may be satisfactorily accomplished by lead 1 alone. The conduction disturbance like those found in the various types of heart block can be well studied in the 3 conventional leads. In actual practice, the coronary group have been responsible for the multiplicity of leads; here, there is no accepted limit in the possible leads that may or should be taken in order to establish a diagnosis. The experience and determination of the examiner still remains the only guide in any doubtful situation and repeated examination may be necessary to demonstrate the pathology, if present.

Some Observations about the T-Wave

Of all of the deflections seen in the electrocardiogram, the T-wave has in recent years assumed a role in clinical medicine which has led many of the older pioneers in this special field of applied laboratory medicine to express alarm at the general implications of misinterpretation of certain changes which take place in this terminal deflection of the electrodynamic cycle. With this in mind, the author quotes liberally from his paper which was published in the New York State Journal of Medicine (57:-2659, Aug. 15, 1957).

"Never before in the history of medicine has a simple scratch on a piece of paper wielded more influence over the lives and fortunes of so many people. I am referring to the ubiquitous electrocardiogram produced by a variety

the ventricles. Vector cardiographic studies have shown that the electrodynamic plane represented by the CF lead pattern is anterior to that picked up by the V leads; it is not unusual to find typical ST segmental and T-wave changes in the CF leads which are not shown in the V leads taken over the same point of the thoracic circuit. Figure I demonstrates the graphic records of a 57 year old man with suspected coronary heart disease but with a more or less normal physical examination for the age period; the conventional and unipolar leads are well within normal limits except, perhaps, for a suspicious V-1 and V-2 pattern. The CF leads, however, show inverted T-waves from CF-1 through CF-6. Parallel chest leads studies are indicated whenever there is sufficient clinical evidence to suggest pathology in the anterior aspects of the heart which are not shown in the routine V lead examination.

It may be necessary in borderline and doubtful cases to continue electrodynamic study with the exploring lead. The esophageal leads are not difficult to take with simple additional equipment; small lesions in the posterior aspects of the heart may be demonstrated in cases with otherwise normal leads. The posterior chest leads used in the author's original 9 lead hookup in 1934 and modified by the Valley Forge Heart Institute Committee in 1948, may also yield objective evidence of certain posterior lesions not discovered by any other electrocardiographic method.

In summary, it may be said that the number of leads required in any given electrocardiographic examination will depend chiefly upon the scope and intent of the study;

What are basic facts about T waves? We are not concerned here with the conduction disturbances or with the various irregularities of rhythm; we will not touch on the characteristic electrodynamic changes which develop as a result of acute coronary occlusion. These are all well integrated in the clinical syndromes associated with these conditions. We wish to discuss only the tendency to consider alterations in the T wave as an entity self-sufficient unto itself. We will also have a word to say about the ST segment under certain circumstances.

What is the T wave? The T wave, in contrast to all other components of the electrodynamic cycle, still offers challenge in regard to its mode of origin. Most authors accept the theory of repolarization following the termination of the excitatory process in the ventricular myocardium. Unlike the rapid depolarization phenomena associated with the spread of the electric gradient over the specialized pathways in the heart muscle and represented by the QRS complex, the development of the T wave is predicated on the summation of an infinite number of electrolytic adjustments to constantly changing differences of potential both within and without the cell membrane. Each cell apparently has a different gradient of electrochemical restoration [illegible]

[illegible] vector predominance. Much has been written recently about the problem of electrolyte balance in the heart muscle. Wuhrmann has advanced our original concept of myocardosis to include the various chemical and toxic disturbances of the myocardium. Minor variations in the diphasic gradient of the repolarization vectors are

of direct-writing electronic machines with special attention to the terminal ventricular complex which Libman once called the "Frankenstein T wave." It seems incredible that in the space of less than thirty years the T wave has grown from a laboratory observation to its present dominant role in clinical medicine. Minute changes in its size, shape, and polarity have become the focus of such scholarly and scientific interest that the fate of individuals and perhaps of nations is dependent in no little part on the interpretation of these alterations. Iatrogenic heart disease has long been recognized, but a more malignant form of this syndrome, spawned by electrocardiographic concepts, is now exacting a frightening toll.

How has it come to pass that this terminal deflection in the electrocardiogram has grown to such stature? How is it possible for a simple directional change in a record made by a laboratory instrument to carry such a burden of clinical responsibility? What are the factors and circumstances which have built up this modern Frankenstein oracle, before which the family physician as well as those occupying high hospital and university appointments must bow with deference and submissive humility? Why does it require so much energy and individual heroism to stand up and engage in almost lethal professional combat in pitting experience and considered clinical judgment against the accepted sanctity of a T wave? In short, just what are the facts about the sinister clinical significance of the so-called abnormalities of this T wave?

No one would seriously question the place of the electrocardiogram in the diagnosis of the various cardiac disabilities.

Can the T wave become grossly abnormal in configuration as result of benign factors? It has long been known that simple vagal stimulation may cause remarkable alterations in the terminal ventricular complex in certain responsive subjects; Scott and Reed produced abnormal T waves by carotid sinus pressure. Morton and his associates showed similar findings in resection of the vagus nerve. Lepeschkin showed the interrelationship between hiccups and such electrocardiographic changes. Melchelke and Meitner using sympathomimetic drugs, produced vagotonic reactions which were reflected in the T wave. Peter also used sympathicolytic substances in determining the responsiveness of the neurovegetative system in relation to the electrocardiogram.

The classic iced water experiment of Wilson and Finch was recently repeated by Dowling and Hellerstein. They concluded that the T wave negativity produced by the drinking of moderately large amounts of cold liquids was due to a delay in repolarization of the posterior wall of the heart. The effects of cold on the heart have been widely studied because of this T wave responsiveness. A military team of investigators has reported a series of studies made under arctic conditions of long exposure to sub-zero environment; here again T wave abnormalities were noted. Of considerable interest in this connection has been the development of hypothermic anesthesia with particular reference to cardiac surgery. Berne has shown T wave negativity at such lowered body temperatures. Lange, Weiner, and Gold have confirmed these changes in experimental animals. Finally, Lepeschkin points out that intra-

promptly reflected in contour and directional changes in the terminal ventricular complex. The T wave is thus apparently more responsive to disturbances of myocardial physiology than any other component of the electrodynamic cycle.

Before reviewing the various conditions in which the T wave may become "abnormal," a working definition of the normal electrocardiogram with special attention to the terminal ventricular complex is of course necessary. Kossmann, in a recent discussion of the normal electrocardiogram, points out that the distinction must be made between the normal heart and the "so-called normal electrocardiogram." Most electrocardiographers accept the concept that the T wave is normally upright in all leads except aVR and perhaps V_1 and V_2 in young people; T_3 may also be inverted in certain individuals. However, the official criteria of the New York Heart Association broaden the base of the list of T wave directional changes which may be considered normal; leads I, III, aVI, aVF, V_1, V_2, V_3, and V_4, may show such alterations in normal subjects. Graybiel, McFarland, and Gates found abnormal T waves in healthy young aviators; I reported similar findings in naval and marine corps personnel. In brief, it is possible, therefore, to find both minor and completely "abnormal" changes in the T waves in eight of the routine twelve leads in healthy individuals; these changes may occur in one or multiple leads. They do not represent heart disease, and they cannot be interpreted in terms of cardiac disability; they simply represent an individual electrocardiographic pattern of a given heart and are thus normal within the framework of specific definition.

have pointed out that the frequency of T wave changes among other electrocardiographic alterations. In 1936 I reported such T wave abnormalities in persons with so-called "chickenbreast" orthopedic deformities.

These are the T wave changes which may be found in normal individuals under varying conditions of ordinary life stress. The list is by no means complete. Taken as a group they represent abnormalities of the terminal ventricular complex which may have no pathologic counterpart, they are abnormal only by definition. It is here that the mythologic sanctity of the upright T wave exerts its malicious sphere of influence, and it is here that the inverted T wave, taken out of context and raised to an exalted clinical entity, has been responsible for so many broken lives and unnecessary financial burdens. I am thinking now of a well known ballet dancer who gave up a brilliant career because of T negativity which I sincerely believe was due entirely to vagal effects; a baseball player with T_1, aV_1, and V_1, V_2, and V_3 T waves changes which were found on physical examination after a heavy meal; a radio announcer with T_1 inverted because of anxiety neurosis; an industrialist who retired at the high of an expansion program involving the welfare of thousands of employees and millions of dollars because an insurance examination revealed certain T wave changes due to a deformity of the chest.

On the other side of this picture are the multitude of unsubstantiated insurance and industrial claims of cardiac disability based in part or entirely on T wave changes. I know of an instance where a man has been collecting permanent insurance disability for nearly 22 years because

venous injection of cold solutions may be productive of many T wave abnormalities.

Eating as well as drinking may cause T wave changes. Post-prandial alterations in the electrocardiogram have been known for years. In 1933 Gardberg and Olsen showed that heavy meals, presumably by vagal reflex action, could produce remarkable T wave changes. Simonson, Alexander, and Henschel confirmed this finding. Rochlin and Edwards and more recently Levit and Dinman have demonstrated T wave changes in single and multiple leads after the ingestion of various amounts of food.

The psychosomatic aspects of T wave alterations also have been the focus of recent investigation. Rørvik and Aarstrand have demonstrated T wave negativity in patients with anxiety neurosis. Ljung noted similar changes which he called the "fright electrocardiogram." Magendantz and Shortsleeve warned about making a diagnosis of heart disease on the sole evidence of such abnormalities in young patients with anxiety neurosis. Plice and Pfister noted many T wave changes in a large group of mentally ill patients. They postulate autonomic nervous system imbalance.

Simple orthostatic factors may produce abnormalities of the T wave. Peterson found that certain subjects, on assuming a standing position after resting in a horizontal plane for more than 10 minutes, may completely invert the T waves in several leads. Chardon and Gross noted similar findings in experimental dogs.

Finally, a word about simple deformities of the chest. The most common of these is sternal depression. Here, Botelho, Medeiros, and Amorin, in a review of the subject,

have pointed out that the frequency of T wave changes among other electrocardiographic alterations. In 1936 I reported such T wave abnormalities in persons with so-called "chickenbreast" orthopedic deformities.

These are the T wave changes which may be found in normal individuals under varying conditions of ordinary life stress. The list is by no means complete. Taken as a group they represent abnormalities of the terminal ventricular complex which may have no pathologic counterpart; they are abnormal only by definition. It is here that the mythologic sanctity of the upright T wave exerts its malicious sphere of influence, and it is here that the inverted T wave, taken out of context and raised to an exalted clinical entity, has been responsible for so many broken lives and unnecessary financial burdens. I am thinking now of a well known ballet dancer who gave up a brilliant career because of T negativity which I sincerely believe was due entirely to vagal effects; a baseball player with T_1, aV_1, and V_1, V_2, and V_3 T waves changes which were found on physical examination after a heavy meal, a radio announcer with T_1 inverted because of anxiety neurosis; an industrialist who retired at the high of an expansion program involving the welfare of thousands of employees and millions of dollars because an insurance examination revealed certain T wave changes due to a deformity of the chest.

On the other side of this picture are the multitude of unsubstantiated insurance and industrial claims of cardiac disability based in part or entirely on T wave changes. I know of an instance where a man has been collecting permanent insurance disability for nearly 22 years because

he has a flattened T wave in lead 1. It all started because of a gastrointestinal upset caused by overeating and drinking. In 1934 a diagnosis of "acute indigestion" was equivalent to acute coronary thrombosis in certain medical groups. The finding of the minor T wave change clinched the diagnosis, and although he has never shown any objective evidence of heart disease in all the subsequent years, he is still labeled as a coronary beneficiary. This type of case is familiar to every physician engaged in insurance or industrial medicine; claims will be paid just so long as "experts" are willing to testify that such T wave abnormalities are caused by heart disease.

We are not concerned with T wave changes which are caused by or associated with objective cardiovascular pathology, although there is a considerable clinical experience to suggest that even here in certain instances the T wave abnormalities may have nothing to do with the predominant cardiac lesion. A woman with mitral stenosis on occasion showed T_1 inversion which subsequently was found to be the result of an anxiety neurosis; she did not have the additional burden of coronary disease. The importance of clinical electrocardiography is acknowledged, but the undue emphasis placed on the pseudoclinical sanctity of the upright T wave must be challenged by those who share the responsibility of preventing the entire concept of the electrocardiographic interpretation of heart disease from becoming a mockery both in the clinic and in the courts of law."

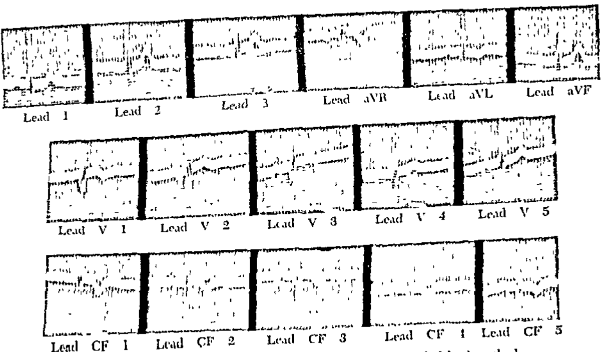


Fig 1 Myocardial infarct localization by the parallel precordial lead method.

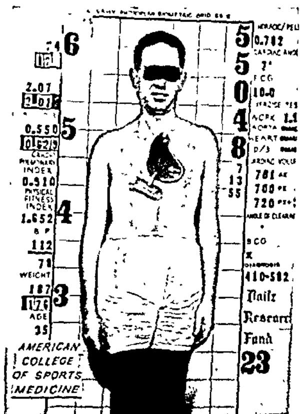


Fig. 2. Normal subject studied by the Alsahy Photoplan Biometric Grid method. Data from his cardiovascular examination are shown on lateral panels A cardiomoulage is made from the teleoroentgenogram or orthodiagraphic x-ray tracing, the third diameter is placed below cardiac silhouette for comparison. After the exercise test, the outline of the heart is traced on moulage with an arrow indicating direction of change, here the arrow points inward showing that the heart became smaller. A dotted white line indicates direction of change after the 5 minute resting period.

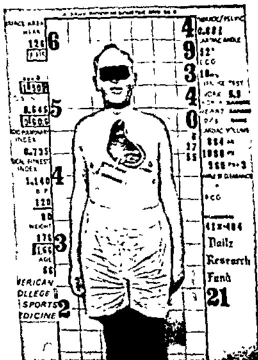


Fig 3 Post-exercise enlargement of the heart in coronary artery

obtained

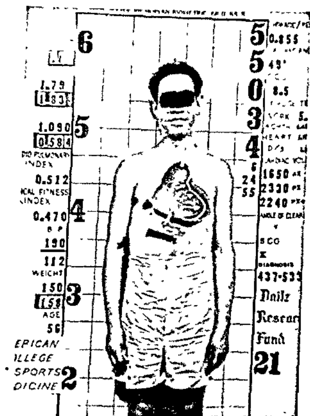


Fig 4 Patient with longstanding rheumatic valvular heart disease; note that Cardio-Body Index is 1 090 compared to the normal for this group of 0 584 The resting heart size is thus about twice as large as normal After the exercise test, it increased to 2330 or about three times that of the normal 780 After the resting period it only returned to 2240. Note the poor Cardio-Pulmonary Index of 0.512.

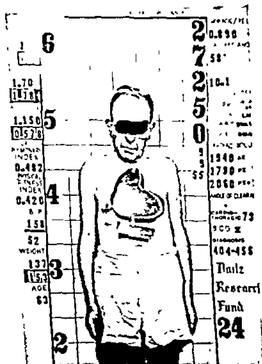


Fig 5 Extremely enlarged hearts may retain some measure of functional capacity The subject here is 63 years old with a long

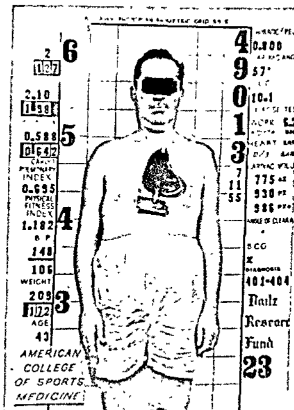


Fig. 6. The "telltale bulge" of early aneurysmal pathology of the left ventricular wall discovered after the exercise tolerance test. The patient had a coronary episode 2 years previously with an uneventful recovery and with a return of his hypertensive syndrome. Note overall increase in heart size from 775 to 930 with the principle enlargement in the apical area and a continued dilatation to 986 after the 5 minute resting period

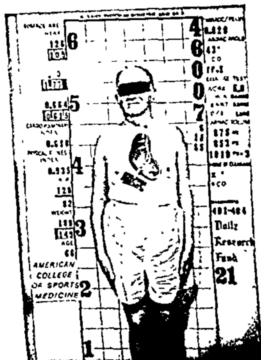


Fig 7 Post-exercise increase in width of aorta. Note widening of descending portion of the aorta from 72 mm to 95 mm, the heart volume also increased from 875 to 1010 Like the "telltale bulge" in early ventricular aneurysms, the tendency toward the development of early aneurysmal change in the aorta may be demonstrated after the exercise tolerance test in certain patients with angina pectoris of the non-responsive nitroglycerine group

CHAPTER VI

PERIPHERAL VASCULAR EXAMINATION

No cardiac examination can be considered complete without some observation of the vascular tree; with emphasis upon the heart, there is a natural tendency to overlook the peripheral vascular system unless the patient specifically complains of symptoms. In the middle and older age groups, disturbances of the coronary arterial tree may have counterpart pathology in the other arterial systems throughout the body; the brain, kidney and legs may bear the brunt of changed circulatory function in a similar or unequal degree. Special tests to determine such disturbances of the cerebral, renal, circulation, and kidney are beyond the scope of this presentation, but study of the vessels of the legs and sometimes the arms must be considered in relation to the cardiac disability present.

The important points involved in inspection of the legs has already been discussed, it may be repeated here that the obvious signs of peripheral vascular disease like color and temperature changes of the toes and feet should be followed by a more thorough examination. Color changes may be roughly grouped into two classes; those which run the spectrum from a pale ghastly white to a deep purple wine color and those which show no or little color

change during the postural tests. With the subject lying flat, both legs are held upright by the examiner or assistant for 3 to 4 minutes, the color of the dorsum of the foot and sole are noted as well as the temperature. The patient then stands upright and color changes are noted and timed; usually one side regains its original standing color before the other or only one foot may be involved. In typical cases, the foot and lower leg may pass from white, through various shades of pink, light red, dusky red, purple and deep cyanotic blue. This group, in spite of the ominous color changes which may be frightening to the patient, usually has a better therapeutic outlook than those with no or very slight color change. In this latter group, the obstruction to arterial flow is usually due to structural changes which do not respond to either medical or physical therapy.

Temperature changes can be determined by running the back of the examiner's hand lightly over the skin areas; sometimes, a distinct line of demarcation between warm and cold areas can be determined which may have no special relationship with the extent of color change. As a rule, however, the deeper the color change, the more likely will be a sensation of cold to the examiner's hand. The patient's own localization of the cold demarcation may differ considerably from that determined by the examiner; this observation may indicate that the process has been a chronic one. Instruments are available for measurement of the temperature changes; thermocouples with a registration accuracy of 0.05° F. are useful in mapping out the chief areas of functional disturbance. If the physician has a sufficiently large number of such patients, the

instrument may be a valuable acquisition in recording the results of treatment; the specific action of certain drugs may be readily noted by temperature changes that are far too small to be noted by the back of the hand method.

Palpation of the dorsalis pedis artery may require some training; the vessel varies in its position from subject to subject and even in the same individual its relation to the bony structures of both feet may be different. Several methods have been described to facilitate this examination; the Samuels method may be useful. The examiner faces the patient's foot and with the thumb and little finger grasping the foot, the index finger tip is inserted in the interbony channel through which the vessel passes. Gentle touching will yield more information than firm pressure. The Protas syndrome should be recognized; in this situation the examiner confuses his own finger-tip pulsation with that of the patient. If the two rates are within the same range of frequency, the Witkin maneuver is suggested; while the index finger of one hand is on the dorsalis pedis of the patient's foot, the other index finger is placed on the radical artery of the examining hand and the two pulsations are compared. It might seem unnecessary to dwell on such details but one of the common diagnostic errors made in peripheral vascular clinics is concerned with the examination of the dorsalis pedis artery.

The posterior tibial artery is less difficult to approach and its pulsations can ordinarily be established with simple finger tip palpation, change in position of the leg may assist in certain cases. If the pulsations cannot be identified in the standing position, the patient should lie prone with the leg relaxed or the knee slightly bent. The femoral

artery in the inguinal region should also be palpated and changes noted.

Pulsations of the peripheral arteries may be studied by a number of instruments; the aneroid oscillometer is a simple and convenient method for investigation of the larger vessels of the arm and leg. Two types of apparatus are available, visual and recording; the visual are magnifying and utilize a moving needle indicator which may be observed like that of a sphygmanometer. In effect, both operate on the same principle of aneroid mechanical amplification. Oscillometers have a gear ratio of 1 to about 57 to 100 compared to the blood pressure gauge of 1 to 4 or 8; in certain patients even the ordinary sphygmanometer may be used. The recording models produce a tracing either by photographic or direct writing methods; the arteriogram is a special form of oscillometric tracing which can also record the pulsations of small vessels like the dorsalis pedis. The oscillogram is chiefly concerned with the height of the arterial pulsations while the arteriogram discloses the form of the pulse beat with its various intrinsic waves.

The objective findings in peripheral vascular disease may show poor correlation with the patient's symptoms of pain, numbness, tingling, cold, heat, or color changes. Complete absence of demonstrable pulsations may produce few or no symptoms while severe disability may occur with minor changes in circulation; not infrequently a patient may complain more bitterly of pain in a "good" leg than that in the opposite side which is more or less pulseless. Wolffe postulates that painful symptoms occur chiefly during the early stages of vascular occlusion; the

chronic phase is associated with a number of compensatory mechanisms which tend to reduce or eliminate the pain factors.

Patients with angina pectoris frequently suffer from intermittent claudication; the leg pains may precede the heart pain by months and sometimes years. They may develop more or less simultaneously in the younger age group but this is uncommon in the elderly. It may be repeated that all patients with suspected or proven coronary artery disease should be examined for peripheral vascular disabilities; there may be an intimate reflex relation between the two syndromes. A recent research report suggests that certain endotoxin metabolites, released into the blood stream when an ischemic muscle is forced to exercise, have a specific constricting effect on the coronary circulation. Nitroglycerin has a special place here; taken *before* the anticipated exercise it may completely suppress the anginal pain with no appreciable effect on the development of the intermittent claudication.

"Night cramps" are not uncommon in peripheral vascular disease; they tend to occur while the patient is quiet in bed and usually after he has been asleep for some hours. The episode is ushered in with a sudden contraction of the posterior muscles of the lower leg in a localized area; the muscle segment becomes rigid and irregularly hardened. With the onset of the contraction, the patient experiences a severe sharp pain not necessarily confined to the area of muscle contraction. After reaching a rapid maximum, the pain gradually recedes and finally disappears, after some few minutes leaving the area with a superficial soreness which may persist for an hour or

longer. Night cramps develop in muscle segments which have become sensitized to a relative hypoxia secondary either to a local ischemia or a general lowering of blood oxygen values. Hypoxia is also responsible for angina pectoris decubitus and the two syndromes occasionally occur together. Rarely both legs are involved in night cramps; the pain pattern is usually repeated in each attack and it may follow a special stimulus which the patient learns to avoid. The most common story is that the patient stretched out his leg or he turned it in a certain position; sleeping with the legs crossed or bent at a peculiar angle has also been noted in such instances. Here the physical examination may reveal only minor objective evidence of vascular disturbance. Subjects with night cramps ordinarily do not suffer from intermittent claudication but this may develop at a later time with a disappearance of the nocturnal syndrome.

Clubbing of the fingers and toes is not usually considered in a discussion of peripheral vascular disease; the condition is seen most frequently in congenital heart disease and in syndromes associated with high grades of cyanosis. Recent studies indicate that a special type of hypoxia which develops at the tips of the extremities is responsible for an increase in the number of venules and a concomitant decrease in arterioles; there is also a relative increase in local tissue substance. Drugs of the sympathetonic group which are therapeutically effective in certain types of peripheral vascular disease are sometimes useful in clubbed fingers and toes when these are producing painful or annoying symptoms like numbness and tingling or hyperesthesia.

Venous Circulation of the Legs

By accepted definition, peripheral vascular disease is concerned only with the arterial side of the distal circulation; the venous return system with its many pathologic conditions has become a separate entity in an age of specialization. In large hospitals, the disease of the arteries of the leg and those of the veins are treated in different clinics; this is unfortunate since a given patient may have both disabilities. There may be and frequently is, a close relationship between the two systems; Buerger's disease, the most disabling of all peripheral vascular syndromes, apparently is initiated by a venous inflammatory process. When the author served as Buerger's assistant in the 1920's, the disease was considered to be a common arterial condition in the Cardiac Clinic of the old Beth David Hospital; the introduction of the antibiotics has within the past decade made true Buerger's disease a comparative rarity.

The relationship between thrombophlebitis and intermittent claudication has interesting sex differentiation; women with more or less extensive venous disease experience far less or no peripheral vascular disability compared to men with similar venous pathology; even in the pre-antibiotic era, Buerger's disease was exceedingly uncommon in women. The deep venous system of the legs should be carefully examined in all patients with coronary insufficiency and leg pains; remarkable relief of the latter condition may occur after a course of penicillin or one of the specific anti-staphylococcus drugs.

Deep venous thrombosis is a frequent cause of embolization; Ornstein's syndrome is characterized by repeated *small infarctions* of the lung from thrombophlebitis. The type and distribution of the rales may be confused with similar findings in congestive heart failure; moreover, when edema of the legs occurs in cardiac decompensation *there is apparently an increased tendency of previous* areas of venous thrombosis to become larger and to release emboli by fragmentation. The persistent type of cough seen in Ornstein's syndrome is, however, not a significant symptom in pulmonary edema due to the failing heart.

Varicose ulcers are a complicating problem in congestive failure; edema of the lower legs tend to aggravate the inflammatory skin lesions and may enhance the patient's discomfort. Removal of the edema fluid by routine diuresis is hampered by the presence of varicose ulceration; if the condition is unilateral, the difference in diuretic effect *may be marked.*

Arteriovenous aneurysms have been mentioned previously; they are more common in the legs and are usually *either* post-traumatic or post-infectious. They may produce no subjective symptoms until an increase in size calls attention to their development. When small, they may be identified by the typical thrill which is imparted by the passage of arterial blood through an irregular opening into an adjacent vein. A characteristic systolic rumble of varying grades of intensity may be heard with the stethoscope over the aneurysmal area; sometimes the rumble or bruit is better heard in the vein some distance from the vessel connection. Arteriovenous aneurysms require prompt sur-

gery; increase in venous pressure may cause right sided heart failure within a relatively short period more especially if mitral disease is present.

Ballistocardiogram

While ballistocardiography is one of the more recently accepted procedures in clinical cardiology, it is chiefly based upon one of the oldest observations in human physiology. Maimonides (1135-1204) described a man whose head nodded with each beat of his heart; Corrigan (1832) in discussing a patient with aortic valvular insufficiency said that the bed shook with each pulsation. Cannon (1916) reported that is was difficult to weigh a certain man on a spring balance scale because the indicator varied as much as 3 pounds with each beat of the heart; the subject had hypertension and Cannon suggested that some clinical use might be made of this finding.

The ballistocardiographic tracing is a record made of the motion transmitted to the body by the movements of the heart and the recoil phenomena associated with this motion. The ballistocardiograph is a relatively simple instrument which is operated through the conventional electrocardiograph. Five major waves or deflections called the H, I, J, K, and L waves are noted, certain smaller waves, M and N are seen in some records. The H wave is probably the result of apical thrust and impact against the chest wall. The I wave is due to the cardiac recoil following the ejection of blood in early ventricular systole. The J wave is probably due to impact of the blood mass

against the aortic and pulmonic arches together with the recoil or deceleration of the blood by both arches. The K wave indicates the deceleration of blood in the descending aorta and impact of the peripheral resistance. The M and N waves appear to be secondary oscillating movements occurring in diastole.

Three general types of body movements are recognized; longitudinal displacement, transverse motion, and rotational effects. Tracings made by various instruments of these specific types of movement may yield different patterns. Considerable debate continues in regard to standardization methods and the type of instrument to be employed.

In the hands of experienced workers in this field, the method may have a more or less clinical value in such cardiovascular conditions as coronary heart disease, hypertension, certain valvular syndromes, and neurocirculatory asthenia or other psychogenic cardiac problems. The ballistocardiograph has also been used successfully in studies of various drugs like alcohol and nitroglycerin, nicotine and smoking, and some vasodilator pharmaceuticals. It can also be used in the exercise tolerance tests.

Ophthalmoscopic Examination

The retinal vessels have been called the peripheral vascular system of the brain. Most cardiologists believe that an ophthalmoscope is an essential instrument in examination of the heart and blood vessel system and ophthalmologists have long boasted that they have diagnosed hypertension in patients before it was discovered

by the internist. The characteristic appearance of the retinal arteries and veins in a number of general conditions is well known and the physician should be familiar with the simple ophthalmic procedures necessary in fundus examination.

Ordinarily, retinal examination does not require local drug preparation; sufficient dilatation of the pupil may be secured by dark room adaptation. For this reason, the ophthalmic study should be made directly after the fluoroscopic examination while the patient is still in the dark room. If this is inexpedient, a 5 minute period of dark adaptation should precede the fundus study at a later and perhaps more convenient time.

Note should first be made of the appearance of the larger vessels; their size, shape, and configuration are important. Special attention should be given to points where an artery crosses a vein for the characteristic finding in hypertension; the light reflex and changes in pulsations are easily seen. Localized spiral or corkscrew appearance of the smaller vessels is common. Arteriosclerotic changes may also be accompanied by various types of exudates and alterations of the retina.

Examination of the eye should also take note of arcus senilis which in relatively young people may be associated with premature degenerative changes in the cardiovascular system.

Exanthema of or near the eyelids is a diagnostic sign in atheromatous disease; it is frequently seen in coronary artery pathology and is common in patients with associated diabetes.

A Final Word on the Cardiovascular Examination

The high points in the objective examination of the heart and blood vessel system have been outlined in the preceding pages; it may not be necessary to perform all of the possible observations on every patient but it may be advisable for the examiner to provide himself with a checklist that will prevent the overlooking of an item which at the moment may not seem to carry any special clinical significance. It has been noted before that the entire examination may require a number of meetings with the patient but treatment should not wait upon the completion of the study. Obvious disability may be discovered in a few moments observation and the most distressing symptoms may be quickly relieved without extensive probing. Time and financial factors may play a not inconsiderable role in such an examination but in complex and debatable situations a successful outcome will be largely predicated upon the accumulation of all the factual data possible.

Laboratory data must be considered an indispensable part of the examination. Urine, blood count, and sedimentation rate are a basic minimum in every cardiac case. Blood chemistries are important in many patients; sugar, non-protein nitrogen, uric acid, cholesterol, and NaCl levels should be determined. In suspected cases of coronary artery disease prothrombin time and transaminase estimations may give emphasis to preventive measures, a concept which will be discussed in detail subsequently.

CHAPTER VII

FUNCTIONAL CAPACITY OF THE HEART IN HEALTH AND DISEASE

The cardiovascular system as a working mechanism presents two separate and distinct clinical appearances both in health and in disease. Although the concept of different entities of the heart at rest and at work has long been recognized, it is only recently that a scientific approach has been made to utilize this observation in the physical examination of the heart and blood vessel system. It has been emphasized before that there may be no correlation between the factual data accumulated during a complete survey of a given patient's cardiovascular system and his ability to perform physical work. None of the definitive data like blood pressure, pulse rate, valvular defects, electrocardiograms, X-ray studies of the heart and aorta, arteriograms, or other objective findings permit an accurate estimation of the subject's actual performance potential. It is well known that patients with advanced heart disease may continue to work at tasks involving considerable physical stress without obvious symptoms; they stand in stark contrast to others with no demonstrable cardiac disability who exhibit dyspnea, palpitation, and sometimes

pain when walking up a single flight of stairs. What is the explanation of this clinical paradox? What is the physiologic mechanism which permits such amazing inconsistencies to develop between anticipated and actual functional capacity? What measures may be taken to close this clinically disturbing gap with all of its unfortunate, embarrassing, disappointing and hazardous implications both to the doctor and the patient?

These are the questions which have plagued internists and more especially cardiologists for many years. This is not an abstract problem; it is one of the most serious situations facing every practitioner who accepts the responsibility of treating any individual with real or suspected heart disease. The specter of sudden death in a patient on his way home from the doctor's office after he and his family have been assured that all was well, is always present to haunt the physician. On the other hand, the man who resigned from high positions and retired into a listless boredom on the earnest advice of his doctor because of serious cardiac problems, does not feel kindly disposed toward the physician after a number of long years have passed by with no change in his physical status; it is possible that other members of his family have since died leaving him in an anomalous position.

Before attempting to enter into any discussion of the problem, it may well be worthwhile to review some of the circumstances which have led to the present situation. Since the turn of the century and with the introduction of specific methods of examining the cardiovascular system, scientific attention and emphasis have been placed

upon the accumulation of factual data; the goal of nearly all research projects has been directed toward more and better diagnostic procedures and for a more complete understanding of disturbances in physiology and biochemistry. Attention has thus been focussed upon the facts of the machine and its parts rather than upon what the machine can or is able to do. Research in performance or functional capacity has been left chiefly in the field of physical education and within the limitations of their professional qualifications, clinical medicine owes much to such capable investigators as Karpovich, Rathbone, Mueller, Massey, Weiss, Brownell and many others. From the goal of physical fitness comes the clue, the author believes, which may help solve the dilemma of miscorrelation between organic heart disease and functional capacity.

Let us start first, by pointing out the fundamental but little appreciated difference between *physical condition* and *physical fitness* when applied to the cardiovascular system. A given man age 50 may be unable to climb a flight of stairs without developing shortness of breath and palpitation, because he is physically unfit. Physical fitness is not a passive, inherent, or natural accompaniment of the body at any age period; no participant in athletic activity would engage in competition without a long and thorough period of training and conditioning; all standards of physical performance are raised by continuous and continued muscular effort. To the man accustomed to a sedentary mode of living, climbing a flight of stairs represents an expenditure of energy which may be beyond his conditioned functional capacity and as a result of which he becomes dyspneic and has palpitation. Work perform-

pain when walking up a single flight of stairs. What is the explanation of this clinical paradox? What is the physiologic mechanism which permits such amazing inconsistencies to develop between anticipated and actual functional capacity? What measures may be taken to close this clinically disturbing gap with all of its unfortunate, embarrassing, disappointing and hazardous implications both to the doctor and the patient?

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functional reserve; since World War I, the importance of tests to determine physical fitness of a normal subject has occupied the attention of the military services. The Schneider Index was developed for the selection of aviation pilots. World War II saw an increase in the variety of tests employed in estimating the functional capacity of the cardiovascular system and the recent expansion of space medicine has added emphasis to such examinations. The application of the methods to patients with heart disease is a more recent trend in cardiology which will assume increasing importance in relation to treatment and prognosis.

The next chapter is devoted to a presentation of the most important function tests; they have been chiefly selected because of their relative simplicity of performance and because they do not require expensive and complicated equipment. Nearly every practitioner possesses all the essential working tools and no elaborate technical methods or assistants are necessary. In actual practice, most of the tests are within the scope of the routine examination procedures employed in the original study of the patient; these procedures represent the basal or resting clinical picture of the patient at rest. A comparison with the results of the same tests after exercise may be startling and no little disconcerning to the examiner. He may have solved, however, by one simple functional capacity test a common clinical situation in which a given patient seeks his physician's care because of pain and dyspnea. After a careful and conscientious examination, abnormalities of the heart or blood vessels have been found and the patient having been reassured, leaves the doctor's office only to

ance in the normal subject thus is dependent upon functional conditioning.

Functional capacity is not automatically lost in heart disease; in patients with previous high physical fitness levels the factors of compensation may permit continued better than average performance levels. Even the heart damaged by myocardial infarction and actual loss of heart muscle substance may retain the capacity for physical conditioning. The traditional concept of prolonged bed rest in certain cardiac disturbances has gradually given way to the belief supported by experience that such long periods of inactivity may do more harm than good in the eventual progress of the disability. The therapeutic implications of exercise in heart disease was pointed out by Sussman over 25 years ago; recent studies have all shown that the heart, as a working organ, requires more than a minimal basic activity to maintain an efficient volume output in relation to metabolic balance.

Assuming that the disabled heart would benefit by exercise and recognizing that physical fitness demands such physical effort, the problem now becomes one of determining what type of cardiovascular pathology is suitable for such measures and which group presents definite contraindications. Experience has shown that not every cardiac case comes within the concept that physical conditioning will improve physical fitness; individuals with low marginal reserve function approach such additional physical stress with a calculated hazard. If this risk is likely to be greater than the anticipated benefits it would be folly to regard exercise as a therapeutic consideration.

A number of methods have been devised to estimate

to supplement the information obtained during the resting examination. The physician is thus urged to select whichever tests seem to be indicated in any given case; he may well be rewarded by the additional data obtained.

return again with the same complaints. What has not been sufficiently recognized in such an incident is that the patient at the time of the examination was not experiencing the complaints for which he came for advice; at the moment when the physician saw him, he had no pain or dyspnea. In fact, he may have felt very well and may have wondered, inwardly, what he was doing in the physician's office at all. On the other hand, if the doctor had an opportunity of examining the man at the time when he was actually experiencing his symptoms of pain and dyspnea, the clinical impression might have been entirely different. The examiner might have found a considerably elevated blood pressure, a rapid and perhaps irregular pulse, marked changes in the electrocardiogram as well as enlargement of the heart by X-ray. In this instance, the heart at rest and under stress presented the two entities previously described, each giving an entirely different aspect of the individual's cardiovascular system. Until the physician has had an opportunity to see both sides of the clinical picture, no considered opinion of the patient's condition is warranted; when either half of the examination is missing only a presumptive diagnosis is scientifically possible, and by the strange twist of fate it is usually the most sinister half which is unknown.

It is not the intent of the author to state that the function tests will completely eliminate the correlation gap beween pathology and functional capacity; many important issues still remain to be investigated but it is not unreasonable to anticipate that the number of cardiovascular casualties may be reduced when such tests are used

cardiovascular disease with many objective findings of disability. These include grossly abnormal ECG's, enlargement of the heart and/or aorta by X-ray, hypertensive heart disease, congestive failure, and a limited capacity for any effort. Such patients are obviously not suitable candidates for any exercise tolerance test since they are unable to carry on with their ordinary daily activities without symptoms. No test is needed to show the low functional capacity which is present. However, some of the physiological tests may be employed in selected cases; as a guide to prognosis and as an index of therapeutic effectiveness, the Cardio-Pulmonary Index, for example, may be determined even in bedridden patients.

GROUP 2 is concerned with subjects showing definitive abnormalities in one or more of the special cardiologic tests but who exhibit a minimum of subjective symptomatology. Such patients invariably desire detailed information about the extent of their potential activities; most of them wish to do more rather than less than the practitioner has already advised. Here, the good judgment and experience of the examiner must play an important part in determining the need for the exercise tolerance tests but it is in this very type of case where the procedure may be most valuable both to the patient and to the physician. The patient's life and welfare may well be dependent upon the doctor's interpretation of the limitations demanded by a given cardiac condition and the responsibility of this interpretation may have many non-medical implications relating to family, social, business, and recreational activities.

GROUP 3 consists chiefly of those individuals with a

CHAPTER VIII

FUNCTION TESTS

Selection of Patients for the Function Test

Not all cardiac patients lend themselves to certain functional capacity examinations; the standard exercise tolerance test involves the climbing of a calculated number of steps. Individuals with unsteady gait or orthopedic disabilities involving the feet or legs are unable to make the test in safety; subjects with poor vision or with easily induced vertigo present special problems which must be recognized. Test steps should have hand rails or other means of support. The hazards of possible accidents which may occur when patients slip or fall while making the test unnecessarily add to the calculated risk inherent in work performance by any individual with cardiovascular disability. Under well controlled conditions, no untoward results have occurred in many thousands of tests; a recent poll in the New York area has not shown a single casualty in the past two years.

Experience has demonstrated that the classification developed by the Cardiovascular Committee of the Daitz Research Fund in 1954 permits full utilization of the exercise tolerance tests with a minimum of clinical problems. Four groups are recognized:

GROUP 1 consists of those patients with advanced

simple clinical procedures. Most of the accepted tests are based upon well known physiologic principles which have been adapted for the demonstration of special aspects of cardiac and pulmonary function. Their use in heart and blood vessel disease pre-supposes that normal pulmonary function is present; diseases of the lungs, bronchi, pleura, and mediastinum which diminish or seriously interfere with the ventilating mechanisms invalidate the tests in whole or in part. The tests approach maximum reliability in heart disease, uncomplicated by primary pathology of the lungs and/or bronchi; pulmonary conditions secondary to cardiac disability, like those seen in congestive failure, do not necessarily decrease the accuracy of the procedures. Tuberculosis, bronchiectasis, emphysema, lung collapse, pleurisy with and without fluid, lung tumors, and certain chest deformities present contraindications; patients with pulmonary edema due to heart failure and hydrothorax, regardless of the fluid levels insofar as they represent objective evidence of the failing heart, may be tested for basal functional capacity.

As previously indicated, the physiologic phenomena associated with the cardiovascular and pulmonary systems lend themselves to mathematical concepts since nearly all of their functions are within the scope of accurate mensuration. On the cardiovascular side, pulse rate and blood pressure are expressed in numerals; on the pulmonary side, vital capacity, breath holding and maximum expiratory pressure or pressure breathing are also determined in specific figures.

The instruments required for the basal physiologic tests are the common working tools of the practitioner; a steth-

minimum of objective pathology but with a long list of subjective complaints; this is a familiar group in every practitioner's office. With few exceptions, such patients present no calculated risk and the exercise tolerance tests may provide the necessary clues in distinguishing between psychogenic syndromes and those based upon actual functional disability. The tests have proven to be of inestimable value in the management of this difficult clinical group which may consist of insurance and industrial compensation claimants with their special motivation problems and their natural tendency to emphasize and perhaps exaggerate their cardiovascular disabilities.

GROUP 4 is concerned with a large miscellaneous classification which includes individuals with concealed heart and blood vessel disease attempting to pass physical examination for one purpose or another; a man, for example, may wish to obtain insurance by denying a cardiac history. An athlete may wish to conceal a heart condition; a laborer may wish to apply for a special job beyond his physical capacity because of heart involvement. Civil service candidates for police, fire, sanitation, or school appointments with unknown or concealed heart disease come within this group. Here, the exercise tolerance and other functional capacity tests may quickly reveal the true status of the cardiovascular condition if pathology is present. In these subjects, the calculated risk is negligible.

The Basal Physiologic Function Tests

The functional capacity of the cardiovascular-pulmonary systems may be estimated at rest by a number of

Vital capacity standing (VC-S) has both relative and bsolute clinical importance. Standard tables based on eight, weight, and sex have been established for normal ndividuals. On the Cardiovascular Work Sheet, a space is provided for the variation from the normal of the patient's given group; the estimation based upon the subject's height compared to weight suggests in a graphic way the effect of overweight in patients who may be reluctant to accept the physician's advice concerning dietary regulation. When, for example, the variation in vital capacity from the normal based on height is 80% and 50% when based on weight, the difference of 30% becomes a strong argument for weight reduction. Non-cardiac sedentary subjects may score 90% while athletes may reach 110 to 120%; scores of less than 85% suggests loss of basal functional reserve.

Vital capacity recumbent (VC-R) is a measurement of the so-called dyspneic factor of posture in cardiac patients. Individuals with heart disease cannot be *comfortable* in a flat position, orthopnea requires several pillows or an inclined head rest. Even in normal subjects, vital capacity is lessened, the average loss is about 10%; thus, if the standing vital capacity is 4000 cc., the recumbent may be 3600 cc. In cardiac patients the loss will usually be greater than 15%; thus, if the VC-S was 2800, the VC-R might be 2100, a loss of 700 or 25%. In marked decompensation, the loss may be as high as 40 to 60%.

Vital capacity in the bending position (VC-B) represents the most difficult breathing posture; the thoracic movements are restricted and the breathing is chiefly diaphragmatic. A rough determination of the relative effi-

oscope, watch, blood pressure apparatus, and a spirometer. Each of the basal tests of pulmonary function requires patient cooperation; the cardiovascular do not depend on the subject. The psychologic implications of the tests will be presented later in this Chapter but it may be said here that the pulmonary data will only be as valuable as the patients desire to give his best performance under the given circumstances.

Vital Capacity Tests

Vital capacity can be determined by a number of methods; the simple spirometer apparatus used in basal metabolism equipment is available in nearly every doctor's office. The Sargeant spirometer, developed at Harvard more than 40 years ago, can be made by the handy practitioner; the water bottle displacement method of Cunningham and Cohen has been in use since 1915. These are relatively large permanently installed types of apparatus. The Saxl flarimeter is a pocket sized instrument for measuring expired air; the Armstrong bellows is a small apparatus and the new Navy plastic balloon method requires a minimum of space.

Vital capacity as a measurement of cardiac functional capacity is determined in three positions. With the subject standing, a deep breath is taken and maximum expiration is recorded. The subject then takes a recumbent horizontal position and, after resting for a few moments, vital capacity is again determined. Finally, in a sitting position with the head and chest bending down as far as possible, vital capacity is measured once more.

mean pressure was 96 mm. Hg. (BP 130/80) and pressure breathing (PB) 24 mm. Hg. the loss is 75%.

Pressure breathing is an index of intrathoracic pressure; when intrathoracic pressure exceeds systolic pressure, no blood leaves the heart. There is thus produced a state of general hypoxia in which the brain reacts with a conditioned responsiveness. Athletes and special test subjects who are able to produce a pressure breathing level much higher than systolic blood pressure without experiencing "blackouts" are better condidates for space medicine activities. While these aspects of pressure breathing are largely confined to the supermen of the physically fit of the nation's youth, a number of clinical implications concerning functional capacity of the heart in health and disease have been discovered and have been found to be useful in cardiac patients.

Giddiness, vertigo, faintness, and syncope occur at or near systolic pressure during pressure breathing in normal non-cardiac subjects; patients with cardiovascular disease experience these symptoms of cerebral reaction at much lower levels. Insofar as hypoxemia is an index of the failing heart the level at which such symptoms occur may represent a clinical index of such changes in functional gas metabolism. The physician thus has a simple but not strictly accurate estimation of oxygen-carbon dioxide relationship which play a basic role in coronary heart disease and in congestive failure.

ciency of the two chief factors in respiration can thus be estimated. VC-B in normal subjects is about 30% less than VC-S; in cardiac patients it may be as much as 50 to 65% less. If the loss is in the normal range, the patient is likely to be a diaphragmatic breather with the implication of some disturbance of the thoracic mechanism. This syndrome will be considered in a later section.

As one of the simple tests of cardiac functional capacity, vital capacity has a definite place in the physical examination of the patient. It may be used without reference to standard tables as a measurement of the individuals response to therapeutic measures; an increase in VC-S and VC-R and a change in their relationship to each other suggests an improvement in functional capacity which, when viewed in its proper perspective may supplement other favorable findings.

Maximum Respiratory Pressure

Pressure breathing represents a number of interesting and complex physiologic problems, many of which are still under world wide investigation in relation to space medicine. The test itself is simple to perform; the subject takes a deep breath and then blows into a pressure measuring apparatus of the type already described. Pressure is determined in mm. Hg. as in blood pressure estimations. Recent studies indicate that there is a relationship between the subject's mean blood pressure and maximum expiratory pressure; in normal individuals the two pressure levels are the same (plus or minus 10%). In cardiac patients, the difference may be in the range of 60 to 85%; thus if the

Cyanosis of the face, lips, and upper chest and neck occur quickly in patients with heart disease. Superficial veins become visable and with red adaptation glasses tiny venules appear in a network pattern particularly over the upper chest areas. There is no special hazard involved in this or the other pulmonary tests. The most frequent subjective complaint is giddiness and occasionally anginal disposed subjects may stop a given test because of anticipated pain but no true attacks have been noted. In this, the basal physiologic tests stand in contrast with the exercise tolerance group where physical effort has produced pain in responsive patients in a number of instances.

Respiratory Index

We turn now to the physiologic index tests where two or more factors are combined in a formula which may have considerable clinical importance both in diagnosis and treatment. The factual data obtained in the basal pulmonary tests and in examination of the cardiovascular system can be expressed in various simple and comprehensive index formulae.

The Respiratory Index of Gross is a well known method of expressing different grades of hidden or nascent dyspnea. When vital capacity is added to the breath holding data, a measurement of such basal dsypnea is obtained; the formula is:

Vital Capacity (in 100 cc) + Breath Holding (in seconds) = Respiratory Index

Thus, an athlete with a VC of 4800 cc. and BH of 120 seconds has a Respiratory Index of 168 while a cardiac pa-

Breath Holding

Breath holding is probably the oldest of physical fitness tests; it was employed by the Greeks in the original Olympic Games in 776 B.C. Breath holding is more responsive to conditioning than vital capacity or pressure breathing; subjects can markedly improve their performance by practice in breath holding. Breath holding skill has received much recent attention in skin diving and also in space medicine; the world's record was made by Jordan in 1956 with 392 seconds (6 minutes, 32 seconds). Young adults and athletes can hold their breath for 120 to 200 seconds.

It is a simple test for cardiac patients to perform; no preparation is necessary. The subject is instructed to take a deep breath and hold it as long as possible; a stop watch is useful but any time piece with a second hand can be employed. This test, like pressure breathing, demands full subject cooperation and a desire to achieve maximum performance. During the test, the examiner should take advantage of auscultating the patient's heart; changes in rate, rhythm, and sound characteristics may be noted as well as the development of gallop rhythm, murmurs, and other sounds not heard otherwise. In normal subjects, the rate first slows down and then slowly increases as the strain factors become greater; the rate at the end of the test is usually 5 to 10 beats faster than at the beginning. In cardiac patients, the rate starts to increase much sooner and the final rate may be 30 to 50 beats faster than the original.

were devised in 1908; these utilized changes in blood pressure. Schneider in 1916 developed tests which included blood pressure and pulse rate before and after a given exercise. The Schneider Index with its standard tables was in wide military use between both World Wars in military aviation. In 1939, Dr. R. Burton Opitz, the well known Professor of Physiology at Columbia University and the author, then a Lieut. Commander, M.C., USNR, working on a project for the Third Naval District in New York, developed a formula which utilized for the first time the pulmonary data in addition to the cardiovascular data of the Schneider Index.

The original H-O (Hyman-Opitz) Index correlated the factual data of the two systems into a formula:

$$\frac{\text{Vital Capacity} + \text{Pressure Breathing} + \text{Breath Holding}}{\text{Systolic Pressure} + \text{Diastolic Pressure} + \text{Pulse Rate}} = \text{H-O Index}$$

Tests on many thousands of normal servicemen and college students showed that the average index was 1.000 (plus or minus 5%). The index was employed in the Medical Department of the Navy during World War II; it had a limited use in servicemen with heart disease.

With the untimely death of Dr. Opitz, the author attempted to convert the H-O Index into a basic formula for clinical use in patients with cardiovascular disabilities. It was soon found that the original H-O Index was valid only in the age groups represented by service personnel from 18 to 28 years. The age factor in the older groups was finally integrated with the pulmonary data and the so-called Hyman Cardio-Pulmonary Index became:

$$\frac{\text{Vital Capacity} + \text{Pressure Breathing} + \text{Breath Holding} + \text{Age}}{\text{Systolic Pressure} + \text{Diastolic Pressure} + \text{Pulse Rate}} = \text{C-P Index}$$

tient *with a VC of 1800 and BH of 35 seconds* has an RI of only 43. Most normal adults have an RI of 120 to 150; compensated cardiac patients average from 80 to 100; scores under 75 indicate a loss in functional capacity.

Improvement in the Respiratory Index occurs promptly in successful treatment; there is a close relationship between effective diuresis, for example, in congestive failure when other clinical findings are not especially striking. A woman age 51 with considerable edema of legs, liver, and the abdominal wall and with rales in both chests from mitral valvular disease had a Respiratory Index of 38 at the beginning mercurial dehydration; 4 days later after a loss of 16 pounds, the index had risen to 60; there was no or little change in heart rate, blood pressure, ECG, or size of the heart by X-ray. With the pocket flarimeter, the entire test is a bedside procedure.

The Cardio-Pulmonary Index

Estimation of basal cardiovascular capacity has been a recognized problem in the military services of all countries for over 100 years; the Imperial German Army in 1846 attempted to develop a method of selecting candidates for special services. It was important then as now to determine which men were able to endure maximum physical hardship without breaking down. Such names as Von Richer, Sturmholz, and Wendt are associated with the so-called Spartan tests of endurance. In England, Bailey developed tests which bear his name, and in this country McKinley, Bates, and Waterson gave the U. S. Army certain tests in 1888. Later, the Crampton tests

rompt clearing of physical and laboratory data with a tationary or only slightly improved C-P Index.

The Cardio-Pulmonary Index should be taken on the nitial examination in all cardiac cases; as a basal measurement of functional capacity it vies in importance with diagnostic data. Together a better perspective is presented to the examiner than either aspect of the physical examination alone, each supplements the other and total clinical appraisal is thereby enhanced.

Other basal functional capacity tests are available; most of these require equipment and training more or less outside the scope of the average physician's practice. Research organizations have the means to investigate a variety of physiologic processes involved in the mechanisms making up the syndrome of heart failure; the Valley Forge Heart Institute and Research Center, for example, can follow through complicated cases with angiographic studies, isotope investigations, gas metabolism problems, and other informative determinations which require highly specialized personnel and equipment. However, the methods already described will ordinarily provide the practitioner with all of the necessary data required for diagnosis and treatment.

Exercise Tolerance Test

The basal functional capacity tests will provide the examiner with an appraisal of the cardiovascular system at rest; equally important but predicated upon the basal data is a study of the heart at *work*. Capacity for work can be determined by cardiovascular reaction to measured phys-

In a large series of normal subjects of all age groups from 18 to 88 the C-P Index gives an average of 1.000. Cardiac patients with minimum disability may score as high as 0.800; in individuals with congestive failure, scores as low as 0.350 will occur. The outlook is poor with scores of 0.300 or less. A typical case is a man aged 54 at the end of the first week after a coronary closure; his clinical data showed:

$$\frac{\text{VC (12)} + \text{PB (34)} + \text{BH (20)} + \text{Age (54)}}{\text{SP (120)} + \text{DP (90)} + \text{PR (88)}} = \frac{120}{298} = 0.402 \text{ C-P Index}$$

About three weeks later, the index was:

$$\frac{\text{VC (22)} + \text{PB (48)} + \text{BH (45)} + \text{Age (54)}}{\text{SP (124)} + \text{DP (80)} + \text{PR (80)}} = \frac{169}{284} = 0.594 \textit{ C-P Index}$$

At the end of 6 weeks the C-P Index had risen to 0.684 and when he returned to his work as a business executive at the end of 5 months the Index had become 0.802.

There frequently is a close correlationship between the C-P Index and the objective data presented by the physical examination, ECG, X-ray, and laboratory studies in patients with heart failure from various causes; in acute coronary episodes, decompensation from valvular or hypertensive disease, and in certain arteriosclerotic syndromes there may be and usually is a parallel improvement in the physical findings and basal functional capacity. This is not necessarily true in patients with persistently abnormal ECG's and other objective findings suggesting no or little improvement after a period of some weeks or even months; here the functional capacity measured by the C-P Index may be much higher than that otherwise estimated. Less common, is the individual with

women, are not likely to be inclined toward sports activities; it has been found that the common denominator in physical effort which is known and practiced by every one is stair climbing. Stair climbing ranks high in the list of energy expenditure activities; in calories per minute per foot/pounds of work performed it is in the same group as swimming, long distance running, and sports like baseball and cricket. In stair climbing, the weight of the patient multiplied by the height in feet climbed within a given period of time equals the foot/pounds of work performed. Thus a 150 pound man, climbing up 20 six inch steps in a given time, performs 1500 foot/pounds of work. Any stairway may be used provided the step height is known and if the examiner is fortunate enough to have such a stairway available it will serve for all of the exercise tolerance tests. Permanent steps in a building are free from the hazards already described in regard to portable steps; they give the patient the security and peace of mind which may be lacking in portable steps.

Most exercise tolerance tests are based upon the performance of 5000 foot/pounds of work: when the patient's weight is divided into 5000 the height in feet to be climbed is determined. Standard tables may be consulted quickly to make the calculation. The Master's tables are also calculated for age groups but it has been found that the comparative age factor may have little significance in most cases On page 185 is given the Daitz Research Fund Tables for all weights from 100 to 225 pounds and for steps varying in height from 5 to 15 inches In actual practice, once having measured the available steps, the only variable factor is the subject's weight; the table may be copied

ical effort; the physiologic response to exercise in normal subjects has been studied by many investigators. Karpovich, Cureton, Jokl, Weiss, and other physical educators interested in work performance and physical fitness tests have established standards of cardiovascular functional capacity in young adults and athletes. With these accepted standards employed as a yardstick, it is possible to estimate work performance in patients with various types of cardiovascular disability.

Fortunately, nearly all of the diagnostic procedures used in physical examination of the cardiac patients may be employed in the exercise tolerance tests; these include the physiologic tests, electrocardiographic studies, and a group of simple observations. Comparison of the resting with the working records presents factual data upon which an adequate appraisal may be made; in general, it may be said that the greater the divergence the more advanced and serious is the cardiac disability likely to be. Normal cardiovascular systems show relatively little change in the various disciplines; the compensatory mechanisms are so well adjusted that the athletes heart at rest and at work shows minimal changes in the special examinations utilized in practical cardiology.

Calculation of Work Performance

Physical stress and effort may be performed in a wide variety of methods; running, jumping, lifting weights, climbing, are but a few of the activities which have been studied. Most cardiac patients, and more especially

CONVERSION TABLE FOR THE CALCULATION OF THE STANDARD 5000 FT/PDS OF WORK BASED ON WEIGHT OF PATIENT AND HEIGHT OF STEPS

Wt. of Pt	No. Ft Cld.	Height of individual steps in inches										
		5″	6″	7″	8″	9″	10″	11″	12″	13″	14″	15″
100	50	120	100	89	75	67	60	55	50	46	43	40
105	47	113	94	81	71	62	55	51	47	43	40	37
110	45	108	90	77	67	60	54	49	45	41	38	36
115	43	103	86	73	64	57	52	47	43	40	37	34
120	42	99	84	72	63	55	50	46	42	39	36	33
125	40	96	80	67	60	53	48	44	40	37	34	32
130	38	91	76	65	57	51	46	42	38	35	33	31
135	37	89	74	63	56	49	44	41	37	34	32	30
140	35	84	70	60	53	47	42	38	35	32	30	28
145	34	81	68	58	51	45	41	37	34	31	29	27
150	33	79	66	57	50	44	40	36	33	32	27	26
155	32	77	64	55	48	43	38	35	32	29	27	26
160	31	74	62	53	47	41	37	34	31	28	26	25
165	30	72	60	51	45	40	36	33	30	27	25	24
170	29	70	58	50	43	39	35	32	29	27	25	24
175	28	67	56	48	42	38	34	31	28	26	24	23
180	28	66	55	47	42	37	33	31	28	26	24	22
185	27	65	54	46	41	36	32	30	27	25	23	21
190	26	62	52	44	39	34	31	28	26	24	22	21
195	25	60	51	43	37	33	30	27	25	23	21	20
200	25	58	50	42	37	33	30	27	25	23	21	20
205	24	57	48	41	36	32	29	26	24	22	20	19
210	24	56	47	38	35	31	28	25	24	22	20	19
215	23	55	46	38	34	30	27	24	23	21	19	18
220	23	54	45	39	34	30	27	24	23	21	19	17
225	22	53	44	37	33	29	26	24	23	20	19	17

Loss in active functional capacity may also be expressed in terms of unit loss per 1000 *foot*/pounds of work performance. Thus, one-fifth of the 29 unit loss would be 5.8

or removed from the book and placed on a convenient wall for ready reference.

The standard 5000 foot/pounds of work may be modified for certain circumstances; patients with easily induced dyspnea or pain should only perform one half of the test or 2500 foot/pounds. Here, one-half of the calculated steps to be climbed should be used. In other instances, a double test is indicated or 10,000 foot/pounds; in athletes 20,000 foot/pounds is sometimes employed. Occasionally, patients are, for one reason or another, unable to climb the calculated number of steps; work performance can then be determined by multiplying the weight by the number of steps climbed by the height of the individual steps.

Respiratory Index Exercise Tolerance Test

The Respiratory Index response to work performance is the simplest test of active functional capacity. After the basal index has been determined from vital capacity and breath holding, the subject is given a 5 minute resting period. He then performs the calculated work on the steps. His vital capacity and breath holding are immediately obtained in that order. Thus, a patient with a basal vital capacity of 3100 cc. and breath holding of 49 seconds has a Respiratory Index of 80; after the test VC is reduced to 2000 and the BH to 31 giving an RI of 51. The reduction is thus 29 index units compared to the original resting or basal index or a loss of about 38%. Normal non-cardiac subjects do not lose more than 10 to 15% so that it may be said that the patient has a rather marked loss of active functional capacity.

dex of 0.402 after the first week of a coronary attack; the index rose to 0 594 three weeks later, and 0.684 at the end of six weeks. He was symptom-free, the ECG's had returned almost to normal. He was walking around and anxious to return to work; he had been climbing stairs to reach an upper level of his home, so that no jeopardy was involved in the standard exercise test. His C-P Index fell to 0 483 after the test; this represented a loss of 0.201 or about 31% or 40 units per 1000 foot/pounds. This was considered to be too great a loss and hence an insufficient restoration of active functional capacity to permit a safe return to his occupation. Five months later, the basal C-P Index was up to 0 802; after the exercise test 0 664, a loss of 138 units or 27.6 units per 1000 *foot/pounds* which is within normal limits of the group.

The correlation between age groups and C-P Index unit expenditure per 1000 foot/pounds of work shows a factor of 0 5084 in several thousand normal subjects of all age groups; the factor is slightly increased in the aged and lessened at age 21. When the patient's age is divided by 2, an approximate standard is determined for his normal energy expenditure measured by the C-P Index. Thus, the 54 year old man quoted above, should have a standard of 27; he actually lost 27.6 which is well within the margin of physiologic error in such data.

As with the Respiratory Index, the progress of therapy can also be estimated in terms of active functional capacity. In a given case of marked congestive failure in hypertensive heart disease with restoration of compensation, the C-P Index unit expenditure of energy was 52 with a normal of 30 in a 60 year old woman; the patient had more

per 1000 foot/pounds of work. The average loss in normal subjects is about 2 units per 1000 foot/pounds.

Respiratory Index Unit Loss (RIUL) is related to oxygen uptake and oxygen debt; the factors of oxygen uptake remaining constant for a given amount of energy expenditure, namely, the 5000 foot/pounds of work, the RIUL becomes a rough measure of physiologic oxygen debt which in heart disease is chiefly responsible for the hypoxial phenomena. Calculation of RIUL or Respiratory Index Unit Loss per 1000 foot/pounds of work performed may be used both in its absolute significance in measurement of the patient's ability to perform work as well as in its relative significance as a measure of his response to therapy. If, for example, this patient with a RIUL of 5.8 was tested a week later after a course of digitalis drugs and diuresis and an RIUL of 4.2 was noted, an improvement in his lessened oxygen debt would indicate a better degree of compensation regardless of other findings.

Cardio-Pulmonary Index Exercise Tolerance Test

The additional factual data employed in the C-P Index permits the factors of oxygen debt to be measured more accurately than with the simple Respiratory Index. The blood pressure and pulse rate changes noted in the old Schneider Index are correlated with the pulmonary factors of the Respiratory Index and the maximum expiratory pressure gradient. The C-P Index determined before and after the 5000 foot/pounds of work test will give significant prognostic information.

Let us take the man previous described with a C-P In-

gists concerning physical stress and the development of changes in the ECG tracing in certain patients.

The most significant changes in the post exercise tracings are the alterations in the pattern of the ST segments and T-waves. The segments tend to be depressed and may drop below the iso-electric line as much as 3 to 5 mm. in certain leads. More evident are the changes in the T-waves; these decrease in height and tend to be flattened and may become completely inverted. Changes in the segments and T-waves frequently occur simultaneously but more often one or the other develops alone. Recent studies have shown that there may be significant differences in interpretation concerning the localization of the solitary change or the dominant alteration when both are present after effort; the discussion is of more than academic interest since the T-wave represents the active phase of the repolarization process and is more responsive to changes in metabolite activity than the ST segment. ST segmental changes may thus represent an insulating loss in the specific transmitting system or an escape from the Purkinje network.

Disturbances in conduction also occur; the P-R interval may be lengthened by 0 02 to 0.05″. More often there is a delay in the QRS complex of 0 02 to 0.06″; occasionally an interventricular block pattern develops and bundle branch block has also been reported. On the other hand, exercise may cause a complete heart block to return to sinus rhythm and there have been instances where previous conduction disturbances have been lessened. In a man age 54 with a P-R interval of 0.28″ the post exercise tracings showed a shortening to 0 20″. Likewise, an interventricular block

or less normal physical findings and slight changes in the ECG and size of the heart. Blood pressure had dropped to 166/100 compared to a previous 246/145; she was rather symptom free and wished to return to her position in the hospital record room. The examiner did not regard this low functional capacity findings to be suitable for her work and a further convalescent period with appropriate medication was recommended. One month later, the C-P Index expenditure was 35 and although there were no other signs of improvement, this was considered sufficient evidence of a working functional capacity.

Electrocardiographic Exercise Tolerance Test

Perhaps the most widely used active functional capacity test is the Master electrocardiographic test; it is limited to determination of relative coronary insufficiency and has little or any application in other types of heart disease. When the coronary circulation is lessened by a number of pathologic processes, the balance between supply and demand may be represented by a very small margin of safety. With an established hypoxia, there may be a constant oxygen debt; a relative ischemia of localized areas of the myocardium may be present and enhanced by any change in lowered blood oxygen levels. Metabolite saturation is a function of oxygen-carbon dioxide concentration and insofar as the repolarization process of the electrodynamic cycle is dependent upon a normal metabolite balance, changes in the electrocardiogram will occur in hypoxia. This, is the current concept among experimental physiolo-

stenographer, had complained that after walking the stairs leading to the court room, she frequently had severe palpitation and shortness of breath. Physical examination including the resting or basal ECG was well within normal limits. After performing the standard 5000 foot/pounds of work test, the tracing revealed atrial fibrillation with a rate of 118, the episode stopped after 20 minutes. A diagnosis of paroxysmal atrial fibrillation was warranted under the circumstances and quinidine was prescribed; unless the exercise ECG test had been made, it is doubtful that the precise diagnosis would have been possible. This case may be compared with another woman age 34 who presented a similar history of palpitation climbing subway stairs; here the post exercise tracings revealed frequent premature beats of the compensated type. While there is no accepted definitive treatment for premature beats, the procaine amid hyprochloride group of drugs are frequently useful, in this instance there was more or less complete relief.

A word may be said here about premature beats for they may be studied in detail during the ECG exercise test. Most authors believe that extrasystoles have minimal clinical significance; they occur in both normal and grossly abnormal hearts. Premature beats are classified in accordance with their point of origin; thus there are atrial, nodal, junctional, and ventricular extrasystoles. Premature beats may also be classified on a functional or hemodynamic basis; extrasystoles which occur very early in the diastolic period are not reflected in the radial pulse. This is due to the pressure gradient mechanism developing in the left ventricle; unless there is a sufficient volume of blood in the

of 0.18″ decreased to 0.12″ after the test. Lengthening in the first cases suggests a hypoxical reaction while in the second a better blood supply as the result of the exercise is postulated.

Changes in the Q-waves have been noted in certain patients with coronary heart disease; no consistent pattern has been observed and correlation studies have not been definitive. Q-waves may increase in amplitude, decrease, and may disappear entirely. Q-waves may develop only after exercise; they may have no relation to other changes in the tracing but they have occurred most frequently in association with marked alterations in the ST segments and T-waves. Significance of the Q-wave changes has been purely conjectural.

Changes in the U-waves have also been noted; the most common finding is an increase in amplitude. They may appear only after the exercise test; when they are large, confusion may result from misinterpretation. The interval between the T-wave and the U-wave frequently suggests an inversion of the T-waves; such records should be carefully studied. Alterations of the U-waves in the post exercise ECG tracings have not received much clinical attention and no importance has been placed on this finding.

Changes in rate and rhythm after the exercise tolerance procedure have already been considered in the physiologic tests; the development of extrasystoles or premature beats was also noted. These changes may be studied in detail by electrocardiographic methods. The type of arrhythmia which develops can be specifically identified and therapeutic indications may be more closely defined. The following case is illustrative: a woman age 36, a court

after exercise in functional coronary insufficiency. There may be a flattening or inversion of the T-wave in this single beat only, with the next sinus beat returning to its normal configuration. Such premature beats may carry important implications concerning the diminished marginal balance in myocardial metabolite balance.

Which leads should be used in the electrocardiographic exercise tolerance tests? The basal or resting tracing should consist of the standard 12-lead hookup; since the post exercise tracing must be taken as soon as possible after the test, only a few leads can be obtained. Many electrodynamic changes disappear within the first 90 seconds after the test is stopped; there must be no delay in connecting up the subject with the apparatus. Personnel should rehearse the procedure until minimum time requirements are met. Some examiners exercise the patients with the electrodes in place; this saves time in re-applying the electrodes but there are certain dangers in the manipulation of the cables which tend to become tangled as the subject turns on the test steps. Mishaps have occurred in awkward individuals which would have been avoided if the patient was allowed to perform the test unincumbered. The suction cup electrodes are easily set in place after the test and are recommended. Most ECG changes are noted in lead 1 but lead 2 may show posterior plane alterations; V-4 is also a useful lead. Many cardiologists use leads 1, 2, 3, and V-4 or CF-4. As a rule, several short leads with a few cardiac cycles each, is more informative than longer and fewer leads; it is worth repeating that the tracings made immediately after the test are the most important.

A third tracing is usually made after a 5 minute resting

ventricle at the time of contraction to raise pressure higher than the diastolic level above the aortic valve, the valve will not open and no blood leaves the heart. Such a premature contraction is known as an *ineffective* extrasystole; when many occur as 10 to 20 a minute, the heart has made this number of contractions without the peripheral circulation receiving the benefit from the work of the abnormal cardiac cycle. Thus, in course of an hour the heart may have made 1200 useless or wasteful contractions; in 24 hours, the number rises to 28,800. Parsonnet called this the "exhaustive premature beat syndrome" which in a heart already depleted by an unfavorable hypoxial balance might spell the difference between recovery or failure. Premature beats, which are developed late enough in diastole to permit a greater filling volume of the left ventricle, will change the pressure gradient sufficiently great to open the valve; a pulse wave then occurs which is transmitted to the peripheral circulation and may be felt at the radial artery. Such transmitted premature beats are always smaller than the normal contraction but the peripheral circulation derives a fractional benefit from the cardiac cycle and there is little loss of function. Premature beats when they occur should thus not be regarded lightly until their functional classification has been determined. A third consideration concerning premature beats, particularly those occurring in coronary heart disease during the ECG exercise test is in relation to the hypoxial effect of the compensatory pause seen in certain very premature extrasystoles. The T-wave of the first normal or sinus beat following the premature beat may show the same pattern changes as that found in the terminal ventricular deflection

after exercise in functional coronary insufficiency. There may be a flattening or inversion of the T-wave in this single beat only, with the next sinus beat returning to its normal configuration. Such premature beats may carry important implications concerning the diminished marginal balance in myocardial metabolite balance.

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period. Occasionally, ECG changes show a delayed appearance; minimal post exercise alterations may become increased during the resting period. If marked ST segmental and T-wave changes have developed in the immediate post exercise records, these changes should be watched; most alterations disappear within the 5 minute period. If these persist, they must be carefully followed by a number of subsequent tracings until the records have returned to the original basal pattern. Burton Opitz pointed out in 1949 that it is the duty of every examiner to observe the patient until the abnormalities developed by the test have disappeared; to do less may be to permit the onset of a coronary episode. If the changes persist for several hours or longer, the assumption that an irreversible lesion has occurred must be considered and appropriate measures taken. Instances have been reported of such patients experiencing severe and even lethal attacks on the way home from the physician's office after a so-called "positive Master test."

There are several debatable aspects of the ECG changes which occur after exercise; some cardiologists believe that some of the changes may be due to other causes than myocardial hypoxia and ischemia. Psychosomatic factors may play a role here as well as orthostatic changes. Not all of the electrodynamic changes are negative in relation to the basal tracing; instead of reversing the polarity of the T-wave, the amplitude of the reflection may become greater. Original T-wave inversions in the basal records may become upright or less negative after exercise. These changes were once thought to represent a better blood supply as the result of the exercise and hence indicated

that the patient should perform more work as a therapeutic measure. More recent concensus puts emphasis on the change alone, regardless of whether the effect of effort increases or decreases the electrodynamic pattern; this concept is based chiefly upon the observation that the normal heart shows no changes except a sinus tachycardia after exercise. Any change in the basic pattern must thus be regarded as evidence of an abnormal metabolite balance; in coronary artery disease this may be the equivalent of a lessened blood supply.

In summary, the exercise tolerance electrocardiographic *functional* capacity tests in carefully selected cases may contribute important factual data concerning the blood supply of the heart muscle in patients with coronary artery disease. Some of the physiologic phenomena associated with the test are not entirely understood and certain ECG findings have not been well integrated into the clinical pattern. Within these limitations, however, the test provides specific information in a group of border line and difficult cases in the degenerative vascular heart disease classification.

Heart Size Exercise Tolerance Test

It has previously been shown that the heart and aorta lend themselves to more or less accurate measurement by X-ray examination; the orthodiagraphic and teleoroentgenographic methods have been discussed in detail. While it is recognized that the "normal heart size" has as many variations as the "normal electrocardiogram," such measurements are of extreme importance when employed in

studying the same heart at rest and after exercise. Inherent errors of a given method may invalidate the absolute size of a subject's heart but will have no appreciable effect in the clinical evaluation of changes which may occur as the result of physical strain or effort.

Normal Physiologic Changes in Heart Size

The normal heart may show marked changes in size and shape following certain physiologic stimulation; the Valsalva test, for example, may decrease total heart volume by 15 to 20% in young athletes. The responsiveness of the heart to this procedure is quickly diminished with loss of functional capacity and it is also decreased with advancing age. The test may thus be used as a simple screening method in all patients examined by X-ray. The basal heart size is first determined at rest and then the Valsalva procedure is employed. Subjects should be instructed how to perform the test and given several trials before the examination is made. If the heart decreases in size during the test, the patient is a suitable candidate for the exercise procedure; if there is no change in heart size, exercise is indicated cautiously.

After the standard 5000 foot/pounds of work have been performed, there is a tendency for the Valsalva effect to be lessened and only slight changes in size are noted in normal adults. In functional insufficiency, there may be no change or the heart may slightly increase in size. After a 5 minute resting period the Valsalva effect returns in the normal heart but it may be delayed for a long period in subfunctional capacity. The test is simple to perform and in

certain cases may be revealing; it may be difficult to attempt in patients with poor cooperation and understanding.

Normal Changes After Exercise

The work of Wolffe, Karpovich, Jokl, and others have shown that the well conditioned heart of an athlete tends to become smaller after exercise; greater volume output demanded by muscular activity is accomplished by more complete emptying of the left ventricle. At rest, the ventricles may only expel 60% or less of the total volume; during work performance about 92% is forced from the heart. Many factors are involved in ventricular output; one of these is diastolic pressure. When diastolic pressure exceeds ventricular pressure, the aortic valves close and the amount of blood left in the left ventricle is known as the *sub-diastolic pool.* In athletes, this pool is used to increase output without increasing rate; increase in rate is usually associated with increase in size. In most well conditioned athletes the heart becomes smaller; this is seen in both normal sized hearts and in those with intrinsic enlargement due to continuous physical activity. This finding is in contrast to the general enlargement which occurs after exercise in patients with various types of heart disease. This post exercise enlargement can thus be used as an index of functional capacity.

In the normal subject, enlargement may occur but it is usually less than 5% of the total heart size determined by the 3-dimensional method. Surface area measurements are unreliable in the effort test since most increases in size ap-

pear to be in the lateral or P-A diameter; the surface area may remain relatively unchanged. The use of the third diameter is emphasized here, since many examiners have reported that no change is usually found in exercise tests when based on the surface area determinations.

The details of the test are simple; after the basal size has been found, the patient performs the standard 5000 foot/pounds of work as in previous examinations. Heart and aortic size are immediately determined and the patient is given a 5 minute resting period after which a third examination is made. At the end of 5 minutes, the heart should have returned to its original size.

In Figure II is shown a wellknown athlete photograph in front of the Alsahy Photoplan Biometric Grid. The grid is used by the Cardiovascular Research Committee of the American College of Sports Medicine in an international study of athletes; it has been adapted here for clinical use in the examination of cardiac patients. On both sides of the subject are placed significant data concerning his cardiovascular examination. In this case, for example, the Cardio-Body Index is 0.550 compared to the normal for this group of 0.629; this indicates that his heart is smaller than average for his height, weight, age, and sex classification. His Cardio-Pulmonary Index is 0.910 which is slightly less than normal for athletes and suggests increasing importance of the age factor which is 35. The blood pressure is 112/78; he weighs 187 against a normal of 176. At the upper right of the grid is the Thoracic-Pelvic Index of 0.782. The anatomic angle is 47° and the ECG is normal (coded from the classification of the American Heart Association). Of special significance here, is the

total size of the heart before exercise of 781 cc.; immediately after the standard exercise test it decreases to 700 cc. and after 5 minutes it returns only to 720 cc. As the subject stands before the grid his height is seen to be 6 feet, 3 inches. The cardiac silhouette has been cut from a transferred orthodiagram or teleoroentgenogram and placed in anatomic position in order to show relationship. On the silhouette is marked the size of the heart after exercise with an arrow indicating the direction of change which, here, is inward indicating that the heart became smaller. Below the heart is attached the third dimension which also became smaller. Studies of this type show at a glance the data accumulated after many hours of examination; the original use, as indicated before, was in connection with a world wide survey of physical fitness. It may be repeated again that until normal standards have been established, the cardiovascular examination of patients with heart disease is limited to known clinical impressions.

It is not the author's intent to suggest that the Alsaby cardiac grid is a necessary procedure in the practitioner's office since it demands more time and equipment than he may afford or be willing to devote to a single case but it is shown here in order to demonstrate the changes which take place in heart disease in regard to heart size after exercise. In Figure III, for example, is a 56 year old man who experienced a coronary occlusion 2 years before; the various data can be noted but attention is called to the increase in heart size from 864 cc. to 1000 cc. after the effort test and a return to 960 cc. after the resting period. This man is obviously not a candidate for the activity demanded in his former work as a building contractor.

Great increase in cardiac size also occurs in patients with already greatly enlarged hearts; the increase after exercise may be very considerable. In Figure IV is a 56 year old man with aortic and mitral valvular disease who has had several episodes of congestive failure; at the time that this study was made he was rather well compensated. The total size of the heart before exercise was 1650 cc.; after exercise 2330, and after the 5 minute resting period it still remained at 2240. Note that the Cardio-Body Index is 1.090 compared to normal of 0.584; in other words, the resting heart is about twice its normal size and after exercise it increases to about three times the normal.

Huge hearts do not necessarily indicate a poor prognosis provided there is retained the capacity to maintain heart size after the exercise test; in certain rare instances there may be even a slight reduction. Figure V shows such a case; here a man of 63 was known to have a markedly enlarged heart for about 22 years. The heart size resting was 1940 cc.; after exercise it decreased to 1780; after the resting period it became larger than the original size 2060. This was due to the secondary dilatation which frequently occurs in left ventricular strain. This study was made in 1955; he died 2 years later from a cerebral accident.

Post Exercise Aneurysmal Dilatation

In addition to the determination of functional capacity measured by change in heart size after physical effort, several incidental findings may develop which could have considerable clinical importance. The chief of these is the discovery of aneurysmal dilatation of the left ventricular

wall. As noted in the discussion of paradoxical pulsation, myocardial infarcts which occur at or near the left profile of the left ventricle may be seen on fluoroscopic or kymographic study. Following the replacement of the heart muscle segments, which have been destroyed in the arterial occlusive process, by scar tissue, ventricular systole produces an outward movement instead of the normal inward in such an area. Development of ventricular aneurysms occur within 18 months to 3 or 4 years after the coronary episode in certain cases. They may, however, be discovered much sooner after the exercise test provided the infarcted area is at or near the apical area of the heart so that its profile may be seen by X-ray examination.

Figure VI shows a man age 43 with a history of coronary occlusion about 2 years before; he made a satisfactory recovery but only has a Cardio-Pulmonary Index of 0 695. He has a hypertension of 148/106; Cardio-Body Index is 0.642 compared to a normal of 0.588 indicating a moderate increase in heart size. Total heart size before exercise was 775 cc.; after exercise 930, after the 5 minute rest period it became larger, 986. At the apex area a localized dilatation developed at the original site of the myocardial infarct. This "telltale bulge" persisted for over 30 minutes and then disappeared; it was probably responsible for the *greater* total heart size after the 5 minute rest period than directly after exercise.

The discovery of the "telltale bulge" has a number of clinical implications. First is the determination of a developing aneurysmal process in the ventricular wall before it can be diagnosed by any other method and the adoption of all possible measures to delay its poor prognosis. Treat-

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POST EXERCISE AORTIC ENLARGEMENT

While the X-ray exercise test has been largely directed toward the changes which take place in heart size, alterations in the visible portions of the aorta may also be discovered. In normal subjects no change in the width of the aortic arch occurs after physical effort; in patients with primary heart disease considerable enlargement of total cardiac size is not accompanied by aortic widening. Aortitus due to both luetic and atherosclerotic disease may, however, show considerable marked widening of the arch and descending portion after the test.

Such a patient is shown in Figure VII; he is a man of 66 with a long history of substernal pain after effort, not relieved by nitroglycerin; there is some increase in heart size with a Cardio-Body Index of 0.664 against a normal of 0.615. The ECG's indicate a moderate grade of coronary insufficiency after the Master test. Here the width of the heart before exercise was 7.2 cm. and 9.5 cm. after the test.

Demonstration of post exercise aortic enlargement has thus been suggested in the attempt to distinguish between heart pain which responds to nitroglycerin and heart pain which does not; the first is considered to be due to acute coronary insufficiency and the latter to certain types of aortitis. Forty years ago, angina pectoris was the subject of heated debate of "coronarians" led by Allbutt and the "aortitians" championed by McKenzie, concerning the mechanism of the syndrome; recent concepts consider that classical angina pectoris, with its *typical radiation* pattern

ment of ventricular aneurysms after they have fully developed presents a difficult problem which requires a surgical approach for definitive correction; in the early stages, however, and if they can be discovered soon enough, therapy directed toward reduction in blood pressure and restriction of the patient's activities may delay or even prevent further progress of the pathology. It is possible also that early surgery in these cases might be useful.

The telltale bulge also supplies information about a previous myocardial infarction. In the industrial and insurance world not all individuals tell the truth, or let us be charitable and say, the whole truth, concerning their medical history. Patients who experience a coronary closure and myocardial infarction may make a complete recovery and may show no signs of any kind of the episode; the group is not large but they often become enthusiastic candidates for new or increased insurance coverage and for positions which may require a normal physical examination and where knowledge of a previous heart attack would definitely limit their qualifications. In such instances, the discovery of early aneurysmal change in the heart muscle wall after the exercise test would be presumptive if not conclusive evidence of a former myocardial infarction. No other clinical condition, except perhaps, a stab wound of the heart or similar injury would be likely to produce such a finding; in the latter condition, surgical scars on the chest wall would be supporting evidence of the non-coronary etiology.

place in the practitioner's diagnostic armamentarium; In certain difficult cases it may add the necessary word in comprehension of the situation.

Other Tests of Functional Capacity

In addition to the physiologic, electrocardiographic, and X-ray methods of determining functional capacity of the cardiovascular system, there are a number of other tests which may be employed. Nearly all of these require special equipment and training which may be beyond the scope of the doctor's practice or need. However, they all have a place in estimating functional capacity and in selected cases, where the other methods have not yielded either the necessary or undebatable information which is sought, they should be utilized.

Ballistocardiography has been added to the graphic methods of examining the heart after exercise. In coronary insufficiency and other degenerative conditions of the heart and blood vessels, the ballistocardiographic pattern may exhibit certain characteristic changes. For example, it has been pointed out that the IJ *stroke* is a function of ventricular contractile force and volume output; in athletes this *stroke* may become larger while in coronary disease it may decrease considerably. The JK stroke is normally larger than the IJ stroke but the reverse pattern is sometimes seen after effort in functional disability. Other pattern changes have also been observed; in the hands of experienced workers like Starr, Dock, and Arbeit, correlation factors have beeen recognized in a few cardiovascular syndromes. Until wider acceptance

and its usual prompt response to the nitrite drugs, can be differentiated from the more painful symptoms produced by aortitis and stretching of the serosal nerve network as a result of sudden rise of blood pressure. Insofar as the nitrite drugs may reduce the blood pressure factors, some relief may be obtained by patients with such types of aortitis but in many instances the drugs are without benefit.

It may be possible, therefore, in any given patient with chest pain after effort to distinguish between the syndromes chiefly produced by coronary disease and those caused by the aortic factors. Individuals with true angina pectoris do not show enlargement of the aorta after the exercise test but many exhibit slight to moderate increase in heart size. Differential diagnosis may thus have some bearing upon medication; the iodides have been the traditional drugs of choice in aortitis. Pharmaceutic combinations like Kiophyllin (Searle), Iocapral (Winthrop), Iodo-Niacin (Cole), Calcidin (Abbot) and Lipoiodine (Ciba) have been found to be useful here.

In summary it may be said that X-ray visualization of the heart before and after the standard exercise test may reveal factual data not available by any other method of clinical examination. As a measure of functional capacity it shares in importance with the other methods previously described. The procedure also provides a diagnostic approach to the discovery of early aneurysmal changes in the left ventricular wall, many months and perhaps years before this discovery is possible by other means. Changes in the aorta after effort may also be noted and the findings utilized in treatment. In short, the method deserves a

mentioned here. The physician, if interested, should consult one of the recent publications on the subject which has expanded its clinical usefulness with the development of newer methods. The dicrotic notch has been correlated with functional capacity of the heart; the height of the notch from the base line compared to the height of the main pulse wave is known as the Dicrotic Index. In athletes and normal adults, the Index is low while in coronary disease and other cardiac disabilities it is high. After the exercise test, the Index is lowered in normal subjects while it becomes elevated in heart disease. The Index tends to be high in hypertensive syndromes but is low in aortic insufficiency.

Of considerable interest is the finding of pulsus alternans after the exercise test; this may sometimes be discovered by skillful palpitation of the radial pulse but it is, of course, better seen in the pulse wave recording. Pulsus alternans is a hemodynamic phenomenon which cannot be determined by any other examination. Pulsus alternans has traditionally carried a sinister prognosis; it is seen in severely decompensated patients and in advanced cases of coronary disease. It may disappear but ordinarily the outlook is poor. Its development after the exercise test carries certain implications which equal in importance any of the other changes noted by the various methods already described. Pulsus alternans, after the standard exercise, usually disappears within a few minutes or even seconds, the patient must therefore be promptly examined on completion of the test. Where it lasts for longer periods greater significance has been attached to its development.

of the procedure makes such correlation data more available, ballistocardiography may be regarded as one of the less important methods of determining functional capacity. This statement should not be interpreted to mean that the procedure has no clinical value; it is merely the author's opinion that continued research and further clinical studies are required before full employment of the method can be enjoyed.

ARTERIOGRAMS

Arteriograms are recorded pulse tracings; all of the superficial arteries may be studied. The radial, brachial, femoral, posterior tibial, dorsalis pedis, and temporal arteries may be recorded. Various types of pulse wave recorders are available; they range in simplicity and expense from the glycerin capsule pneumatic system to very accurate electronic strain gauge apparatus. Arteriograms are usually taken simultaneously with the electrocardiogram and phonocardiogram. Arteriograms give factual data concerning the hemodynamic system; the factors involved in arterial pulsation can be carefully studied. In the normal cardiovascular system there is constant correlation between the electrodynamic and hemodynamic systems; in heart disease there may be distinct differences. In the arrhythmias, for example, not all ventricular contractions are accompanied by a transmitted arterial wave; this has been discussed in premature beats and in atrial fibrillation when a pulse deficit occurs. The pulse beat is composed of five distinct waves each with physiologic significance; only the dicrotic notch is

CHAPTER IX

ANGINA PECTORIS, CORONARY INSUFFICENCY, CORONARY OCCLUSION

In a community enlightened by the public health programs concerned with heart disease, the development of chest pain now carries a significance not experienced by any previous generation of anxious patients; all public health propaganda achieves an eventual goal at the expense of certain psychosomatic implications; the development of cancerophobia among susceptibly inclined individuals, while overburdening the cancer detection clinics, has definitely increased the number of patients who have benefitted from early diagnosis and treatment of malignancy. In a similar way, cardiophobia has become a syndrome which the physician must recognize and be able to evaluate in terms of medical expediency. In heart disease, as in cancer, the overtones of anxiety neuroses frequently enhance all the subjective aspects of the condition and since heart pain may be the only symptom of disturbed cardiac physiology, a clinical appreciation of this complaint is of the utmost importance.

All textbooks on cardiology emphasize that chest pain may result from a variety of causes not directly associated

Summary

The various methods of estimation of the functional capacity of the cardiovascular system have been presented in detail to permit the examiner a choice of procedures in correlating the results of the physical examination of the patient with his capacity for physical effort. It is not usually necessary or expedient to utilize all of the tests in anyone case; in doubtful instances, more methods of approach to the problem of determining the extent of an individual's ability to perform work of a given type may have to be made than in others. As an aid to diagnosis, treatment, and prognosis, the function tests occupy a special role in all disabilities of the cardiovascular system. The factual information secured, cannot be obtained by any other method of examination.

Prognosis in heart disease will assume increasing importance as the nation's population grows older and as the degenerative conditions of the heart and blood vessels become a greater social and economic problem as well as medical. Emphasis on cardiac rehabilitation and a prolonged span of industrial usefulness will permit many individuals with disabilities to continue the enjoyment of many additional fruitful years. Progress and full attainment of this goal may be one of the chief responsibilities of the practitioner.

complaints not sufficiently self-impressive to bring him to the physician's attention, the likelihood of coronary dysfunction is very probable. This is particularly true of individuals who have boasted of unusually good health and in whom a show of physical prowess as they have grown older has become a fetish; this applies to the middle aged athlete who reluctantly admits a lessening skill or a loss of functional capacity. When this group confesses to heart pain, the symptom can be accepted at its full evaluation.

The Pain Factor

The differential diagnosis of heart pain is subject to the same limitations as the evaluation of painful stimuli in general; individuals show a wide variation in their subjective response to pain. Every physician is familiar with the clinical gamut of reaction to pain; the clinical pain spectrum extends from the neurotic lady who protests bitterly on the inflation of a blood pressure cuff to the complacent gentleman who suffers minor discomfort with fractured femur. Many of the older physicians recognized the need for a simple scale to measure pain response; Libman developed the mastoid pressure sign; Brooks used the xyphoid reflex; Besser employed hyperextension of the elbow. In 1930, we described a modification of the Rehfisch compression test which was one of the first methods of scientifically estimating the threshold of pain and which is still a simple and informative procedure A blood pressure cuff is applied loosely to the lower leg, unobtrusively and unknown to the patient, a

with the cardiovascular disease; the didactic list includes a large group of functional and pathologic syndromes involving all of the structures within the thorax and upper abdomen. Chief of these are disturbances of the aorta, esophagus, trachea and main bronchi, hiatus hernia, gastric and duodenal disease, gall bladder dysfunction, pancreatic problems, and changes in the colon. The onset of acute abdominal conditions like appendicitis may simulate coronary occlusion. The pain of spontaneous collapse of the lung may be mistaken for severe angina pectoris. Herpes zoster occurring in the left thoracic neural area may be confused with heart pain. The medical literature of the last 30 years is replete with mistaken diagnoses of acute coronary disease; as many as 41 etiological factors have been described in differential diagnosis.

Fortunately, the possible confusion engendered by the very multiplicity of the causes of apparent heart pain is limited by a simple and rapid appraisal of the patient's history and physical examination; in most instances, the practitioner will be able to eliminate all or nearly all of the extracardiac syndromes which simulate coronary dysfunction. The history of previous episodes which were related to one or another of the didactic list or the obvious signs of other intrathoracic or abdominal disease may indicate the probable diagnosis. It must be pointed out here, however, that acute coronary conditions may occur simultaneously with all of the syndromes previously listed but this is not a common experience. Where the episode of heart pain is the first such symptom in a patient who has previously been free from subjective

Localization and distribution of the pain pattern may be important in differential diagnosis; in defining the *precordium* as that part of the thorax overlying the surface area of the heart, it is noteworthy that in a study of 1000 cases of acute coronary occlusion, the localization of the point of maximum intensity was outside this area in more than 61%. The upper part of the midsternum and the left axilla were frequently involved. In contrast, the pain of uncomplicated angina pectoris is usually maximum over the precordial area; further, the secondary radiation phenomena of angina pectoris is much more extensive than in coronary occlusion. Sometimes the radiation pain is more severe than the precordial; occasionally, the radiation pain occurs alone or may persist long after the precordial pain disappears. Many clinicians believe that there is no consistent pain pattern which is characteristic of either angina pectoris or coronary artery occlusion and if either condition is suspected, appropriate treatment should be started at once, regardless of the localization and distribution factors.

The emphasis upon the pain component of the syndrome stems from the fact that this may be and frequently is the predominant symptom observed; it also highlights the need for the prompt relief of the *pain*. Nitroglycerin still remains the most useful drug in distinguishing between simple angina pectoris and acute coronary artery disease; if sublingual nitroglycerin in gr 1/100 (0.75 mg.) doses repeated if necessary for 2 or 3 times *does* not relieve the pain within 5 to 10 minutes, a coronary episode may be suspected. The onset of more *or* less profuse sweating, particularly over the face and upper

rough metallic plate is inserted between the skin area and the cuff. The best results have been obtained from a kitchen vegetable grater about 2 or 3 inches in diameter. The pressure is slowly raised in the cuff and the patient's reaction is quietly noted; in subjects with a low threshold, a startled expression of pain will occur when the pressure reaches 40 mm. Hg. or less. Where the threshold is high, a pressure of 200 to 225 may be reached before the development of complaints; most individuals react at pressures from 100 to 150 mm. Hg. In using the test, the subject must not be aware of its purpose and he must not be told that a painful stimulus is being employed. More recently, the dolor scale of Wolf and others has proved to be of clinical usefulness; the Opitz modification employs a scale from 1 to 10 in which 10 is the memory of the most severe pain ever experienced by the patient. This may have been a toothache, a fracture, or an acute appendicitis; with this in mind, the individual is asked to evaluate his present pain. It is interesting that many patients in the attempt to evaluate their pain on such a scale, will subconsciously diminish the severity of the present episode; for example, they may have complained originally to the physician that the pain is overwhelming or too great to bear but on evaluation they may consider the pain to be only 5 or 6 in the scale. Self-appraisal of the pain may also have certain psychosomatic implications; removing the patient's attention from the subjective to the objective aspects of the pain frequently lessens the anxiety factor and diminishes the vicious cycle of increasing functional incapacity of the involved myocardium, a problem which will be considered later.

Early Management

Where the attack has occurred in a public building or highway, as under XD (extra domiciliary) conditions, the patient should be transferred to a hospital as soon as possible even though the pain has subsided. Except in cases of rapidly developing shock and general prostration, an ambulance is unnecessary; most patients feel more comfortable in a sitting position. Dyspnea and faintness is less in this position than recumbent. The patient should be assisted into the back seat of any automobile and supported in a semireclining position during the trip; at the hospital admission area, he should be put in a wheel chair and taken directly to his assigned bed and adjusted with his chest and head higher than his abdomen and legs. These details of the transfer, although seemingly irrelevant, are emphasized in order to clarify the current opinion in regard to the early management of the acute coronary syndrome; the question of moving the patient, for example, has been the subject of medico-legal controversy. Our experience has shown that no harm results, even in the most serious case, if the removal is made within one hour after the onset; in certain instances such transfer can be accomplished without untoward effects within the first 24 hours. If the patient is seen in XD status there will be no problem about the removal to a hospital; where the episode has occurred in the patient's or someone else's home, the question of immediate removal will depend upon a number of factors. First is the presumptive

chest together with a gray ashen pallor, is typical in coronary occlusion; it is rarely seen in angina pectoris where the patient may have a ruddy face especially when nitroglycerin has been administered. To experienced observers, there is a marked difference in the facies of the two conditions; anxiety and apprehension predominate in the coronary group.

All cardiologists believe that control of the pain factor is the most important consideration in the emergency treatment of a coronary attack; the abnormal reflex physiology produced as the secondary effect of the pain has a tendency to increase the hypoxic phenomena of the syndrome. Experience has shown that prompt amelioration of pain statistically enhances the possibility of ultimate recovery and the reduction of disability. The drugs of choice remain the narcotic group; Demerol, Dilaudid, Pantapon, Papaverin, Codeine; morphine is the least desirable since its vagal effects may have a tendency to increase nausea and vomiting as well as depress blood pressure. Administration is always hypodermic or intramuscular; intravenous injection has certain hazards and should be reserved for special indications later to be discussed. Dosage should be sufficiently large to assure more or less complete narcosis; this point will vary from patient to patient but it has been found that one large initial dose is more effective than 2 or 3 repeated small doses. There is little use here of the sedative drugs; the barbiturates and the recent group of barbiturate substitutes in large enough doses to control pain may produce a number of undesirable side effects.

duration of the disease; this policy may be and frequently is fruitful of acrimonious questioning by the family. The physician may inadvertently make a statement which later is taken out of context and given unwarranted emphasis; this can lead to a series of unfortunate and embarrassing discussions which terminate in greatly disturbing the peace of mind of all concerned. Insofar, as such a situation does nothing to improve the patient's condition, it is a far better policy to be as frank as possible about the length of the disability; after the initial period of dismay has subsided; both the patient and family can now make the adjustments and arrangements necessary under the circumstances. The day to day prognostic method has much to condemn it; it is unfair to the patient, family, business associates, as well as to the physician; an honest appraisal of the disease, given at the very start will subsequently be appreciated by all those who may be concerned with the situation.

POST ANGINAL SYNDROMES

In many instances (about 38%), the patient becomes symptom-free shortly after the initial episode of pain; at the end of the first 24 hour period, he may resent bed-rest and may become difficult to control. If the laboratory data have become positive with significant changes in the ECG's, leukocytosis, increased sedimentation rate, elevation of the transaminase levels, slight temperature, and possible changes in the heart sounds, a definite diagnosis of myocardial infarction is sufficient to restrain the patient's enthusiastic desire to return to his usual routine

seriousness of the attack; it is not necessary to hospitalize every case of coronary occlusion. If the home is well equipped to handle a sick person, most minor episodes can be well managed by family arrangements; where the patient lives in a small apartment or a hotel room and has no immediate family or friends, hospitalization is indicated chiefly for ordinary nursing and care rather than for specialized services. On the other hand, if the myocardial infarct is extensive enough to require oxygen and intravenous shock therapy, hospitalization is definitely demanded. As a general rule it is not advisable to move a patient in cardiogenic shock but it has been done under extenuating circumstances. Visitors to New York or any other large city who experience a coronary attack and are seen by the physician in their hotel room may require removal regardless of the seriousness of the occluison, many of the metropolitan hotels have a more or less well equipped hospital unit in the building for such emergencies and removal of the patient presents little difficulty.

Dr. Harlow Brooks once said that an acute coronary occlusion is a three times 90 day disease; the first 90 days are concerned with the acute manifestations, the second 90 days with the subacute, and the third with the convalescence and return to work. While this time schedule will not fit every case, it presents a practical working management plan; with this in mind, the family and sometimes the patient should be told at the very beginning of treatment what the probable period of disability will be. Some physicians have a tendency to evade the issue by adopting a day to day estimation of the

to the physician in charge of the situation; most episodes of angina pectoris, regardless of the temporary severity of the attack, have a tendency to completely disappear within a period of 15 to 20 minutes. Sometimes, the pain may last in a mild form for about one half hour. As a rule, with the disappearance of the pain, the patient will feel unusually well. Many authors have commented on the euphoria which may follow an anginal episode; several explanations have been offered. The relief from any severe painful stimulus is naturally accompanied by a feeling of well being; the return to *status quo ante* regardless of the disability present, has psychosomatic implications. The euphoria following an anginal attack, however, usually goes beyond the previous *status quo* and the patient may feel better than he has been for several hours or even days previously. The sense of well being may also be accompanied by actual increase of functional capacity; it is not an uncommon experience to note that in performing a given type of physical effort, like walking, that the patient can proceed with his activities without further disability. Many individuals recognize this peculiar pattern of the anginal syndrome from previous attacks and there are instances where patients have reported that they have looked forward to having an episode with the thought in mind that they would subsequently be free to continue with their customary routine without further restriction for the rest of the day.

Cardiac physiologists like Opitz and Karpovich have correlated this clinical observation with the phenomenon of "second wind" in athletes; it is a common experience

manner of living. The problem of explaining a symptom-free condition with significant laboratory findings is not difficult. When, however, there are no such confirmatory tests or when the significance of laboratory data is debatable, management of the case becomes more difficult.

How long should a patient be confined to bedrest under such circumstances? The physician may have to answer this question frequently. At a recent panel discussion, a group of distinguished cardiologists agreed that a minimum of one week's observation was mandatory in such patients with negative or non-confirmatory laboratory data; the development of ECG changes may require 5 to 6 days in certain cases and there may be an associated lag in the demonstration of the important laboratory findings. If no such objective information is obtained, release from bedrest is permissible; there should then follow another week or ten days observation during which the patient becomes more active. A final ECG is required before the individual is given a full program.

Episodes of simple angina pectoris which may be secondary to small areas of subendocardial infarction may produce none of the usual laboratory findings but at the end of a ten days' or two weeks' period may show small but significant ECG changes. It is possible that many instances of so-called severe angina pectoris which become symptom-free in a matter of hours are actually episodes of miliary infarctions which are too small to produce constitutional reactions.

When is the emergency period of heart pain over? This question constantly presents a psychological hazard

curs instead of dilatation. There thus occurs an *actual* diminution in blood volume in the presence of an increased demand; Sussman sees a teleologic significance in this reaction. The augmentation of the factors leading to the pain signal may be life saving in instances where there is a tendency for the reflex mechanism to be lessened.

Nitroglycerin

The therapeutic effectiveness of nitroglycerin in the typical instance of angina pectoris is chiefly due to relaxation of the constrictor reflex in those parts of the arterial tree which are still more or less free from vascular disease; it is doubtful that arteries which have extensive intimal and medial pathology of the type produced by the atheromatous syndrome play any part in the functional dilatational phenomena which are associated with relaxation of the constrictor mechanism. The lessened capacity of the diseased vessels is due to a decrease in the internal diameter; intimal swelling and placque formation with the development of murantic thrombi narrows the lumen in accordance with the hydraulic law of Henderson which states that a 2 inch pipe line has four times the capacity of a 1 inch. In other words, narrowing the vessel to one half its diameter, decreases blood flow to one fourth of its normal capacity. The reverse is also true; doubling the diameter of a vessel, increases its capacity four times. The therapeutic effect of the dilator drugs is based upon this hydrodynamic law.

among medium and long distance runners that an initial period of dyspnea and palpitation and occasionally precordial pain or distress will disappear as a contestant continues with the race. Many athletes depend on this second wind for the final spurt in any close competition. The explanation offered is based on the development of an additional coronary arterial blood supply potentiated by the factors of an increased electrolyte metabolism. Even in the well conditioned athlete, objective signs of functional coronary insufficiency may occur; Wolffe, Jokl, and others have found abnormal ECG's in such runners. These electrodynamic changes exhibit the same pattern as that seen in patients with angina pectoris with ST segmental and T-wave alterations; in certain instances, the abnormalities persist for as long as 2 to 3 hours although most disappear within 10 to 15 minutes after the race.

The difference between the athlete and the patient with angina pectoris is thus apparently a question of coronary artery responsiveness to the demands for increased volume flow; whereas the normal coronary tree has a remarkable potential capacity, diseased vessels rapidly lose this important reflex function. The concept has long been held that when demand exceeds supply, heart pain develops; the factors involved in the supply are usually considered in terms of a slight or no increase in the capacity of the coronary tree. A postulate advanced by Parsonnet challenges this view; there is experimental evidence that in such patients a disturbance in cardiac physiology produces a reversal of the normal reflex mechanism so that a constriction of the vessel oc-

sublingual was originally effective it may require 3 or even 5 such tablets to obtain an equal degree of relief after several months or years of the condition. This phenomenon of tolerance has been utilized in the treatment of new cases who have severe secondary by-effects, if the patient's choice has been deterred by such reactions the dosage should be reduced to one half or even one quarter of the standard 1/100 grain tablet. It requires some dexterity to divide the small nitroglycerin pill into halves or quarters; but since it dissolves quickly in water the necessary fraction is easily obtained. The patient soon learns to balance the relief from his heart pain with the disagreeable secondary reactions; as a rule these reactions become less in each succeeding attack. Long time sufferers from angina frequently remark how difficult it was to take nitroglycerin in the beginning but now they can use several tablets without experiencing the cerebral reactions.

It should be pointed out here that the patient's voluntary increase in the dosage of nitroglycerin in order to obtain an equal relief from his symptoms as time goes on, does not necessarily indicate that his attacks are becoming more severe or that his condition is worsening. This *post hoc* reasoning has been responsible for many instances of undue worry and anxiety on the part of the anginal sufferer as well as the physician. It should be recalled that the commercially available nitroglycerin tablet exhibits a pharmacologic loss of potency in accordance with the factors of time and temperature; if the drug has been carried in the patient's pocket over several months, the loss in tested samples may be as

The use of nitroglycerin in angina pectoris may sometimes be limited by certain secondary reactions. The chief of these is related to the vasomotor responsiveness of the arteries of the face and head; patients may complain of symptoms which vary from a slight flushing of the face to an unbearable burning sensation which may persist for several minutes or even a half hour, followed by *a hypersensitivity of certain skin areas.* Fullness of the head, vertigo, mild headaches, and occasionally a painful throbbing cerebral syndrome may occur. These reactions may discourage the full employment of nitroglycerin in many patients not withstanding the prompt and almost complete relief which they may experience with the drug during an anginal episode. The older clinicians were familiar with these side effects; Mackenzie spoke of the pseudo-transfer effect of the nitrite compounds. By this he meant that the patient's pain was transferred from his chest to his head and the continued use of the drug was dependent upon the individual's choice of which was the lesser disability. Rehfisch is *credited with the play on the word choice; he pointed* out in 1910 that nitroglycerin was truly a drug of choice —choice of pain in the heart or choice of pain in the head! Cabot showed that these reactions are not necessarily unrewarding when considered in terms of the patient's responsiveness to nitroglycerin. Those individuals who have no such secondary reactions are less likely to experience the full therapeutic effect of the drug.

Tolerance to nitroglycerin is common in anginal patients who suffer repeated episodes over a long period of time; where an initial dose of one 1/100 grain tablet

of the episode than give evidence of the condition. A judge in a court room, a performer on television, an executive at a board meeting, a teacher in a class room, all have the common problem of concealing their anguish. Persons under lesser public scrutiny may likewise feel the necessity of not calling attention to their pain. This reticence may have clinical implications; long continued and uninterrupted anginal episodes may pave the way to a myocardial infarction of greater or lesser degree. A number of such instances have been reported; a few have terminated fatally. Any device or gadget which will permit administration of the drug in an unobtrusive manner may thus be of the greatest importance to the patient and he should be encouraged to make full use of them The whole situation may appear to be incongruous and incomprehensible to the average person and sometimes to the physician, but an insight concerning the special problems of such patients has a certain significance in the management of angina pectoris.

Alcohol

Brandy has had a traditional role in the treatment of anginal pain for many generations; from the 1870's until the turn of the century, alcohol in one form or another, was generally employed for the relief of the attack. A sip of brandy permitted to remain in the mouth and under the tongue may frequently be just as *effective* as nitroglycerin in certain patients; the anginal pain of effort seen in elderly men on walking may be quickly relieved by alcohol without the distressing cerebral by-

great as 50 to 65%. A vial of tablets left in the glove compartment of an automobile for only 3 weeks during a summer vacation lost 43%.

In this country, however, nitroglycerin has remained over the years the most frequently used remedy, although a number of new substances have been introduced from time to time. Nitroglycerin has been combined with concentrates of Vitamin A to form a potent and relatively long lasting solution which may be applied to the tip of the tongue with a dropper or tiny glass rod attached to the stopper of a special designed bottle; such a compound has been in use at the Valley Forge Heart Institute and Research Center. A recent innovation introduces a small plastic squeeze ampul which releases a drop or two of spirits of glonoin. An automatic dispenser of nitroglycerin tablets has been attached to a gold pencil and the back of a wrist watch contains a small pill box for the tablets. These are but a few of the methods used in the attempt to make the drug easily accessible. The attention focused on these details stems from the fact that angina pectoris is particularly a disability of men and women who are subject to great emotional and mental stress; as a group they constitute no small part of the high echelon of industrial and management executives; the judiciary, educational, and members of show business also have a high percentage of anginal sufferers. When attacks occur during their public activities, they may be loth to exhibit their disability by taking medicine of any type. There is a natural embarrassment to stop in the midst of the proceedings to take a pill; indeed, many would rather suffer the pain

widely misused than oxygen. Oxygen has a special and definitive role in cardiovascular conditions; it is specific in hypoxia. By definition hypoxia is a state of insufficient oxygen saturation of the blood and body tissues. Hypoxia is the end result of several complex metabolic disturbances; it occurs in a number of clinical syndromes. Insofar as the cardiovascular system is concerned, hypoxia develops whenever the volume output of the heart is lowered beyond a certain critical level. Both forward and backward failure are involved; the combination of lessened systemic circulatory flow and a loss in pulmonary function are the factors chiefly responsible for the appearance of hypoxia. The clinical response to hypoxia is dyspnea but heart pain may also be a manifestation although many patients with extreme orthopnea experience little or no pain. Likewise, but to a lesser degree, individuals with severe heart pain may have no subjective symptoms of dyspnea.

The normal utilization of oxygen is by way of the ventilating function of the lungs, where pulmonary function is markedly decreased by disease or by the accumulation of fluid, oxygen has been administered intravenously. The theoretical hazards of injection of any gas into the circulation are based upon the concept of embolism Accidental air embolism is a well known cause of death, the mechanism is largely due to the accumulation of air in the ventricles and the drop in volume output; the difference between the compressibility of air and the incompressibility of blood as a liquid spells the difference between valves which do not open and those which are forced open by pressure factors. Gases with

effects of the nitrite drugs. As an almost forgotten remedy it still deserves a place in the medical armamentarium for the disease. The historic development of the modern nitroglycerin containers stems from the artful concealment of the brandy flasks of the older generation. A small bottle of brandy carried in the pocket or purse still serves a useful purpose for some patients.

Oxygen

Oxygen may be of considerable value in terminating an anginal attack; until recent years the employment of oxygen has been limited to home and hospital administration. The large tanks and the necessary equipment, whether by tent, mask, or tube, confined its use to indoor areas, although portable equipment has been available in ambulances for some time. The development of small oxygen capsules, no larger than a cigar and containing about 10 liters of the compressed gas, have been found extremely useful in patients with a poor nitroglycerin tolerance. Fitted with a removable mouthpiece, the capsule is easily carried in the pocket; ordinarily 5 to 10 deep inhalations of the gas will give prompt and complete relief. One capsule may provide sufficient oxygen for several anginal attacks. One or more of such capsules may find a place in the doctor's emergency bag for the treatment of this and other conditions as acute pulmonary edema.

A word may well be said at this point in regard to the use of oxygen in general. Parsonnet once commented that no drug in the treatment of heart disease was more

upon a variety of circumstances, the most important of which is, of course, availability. If all are accessible, and if the patient is conscious and rational, it is usually wise to permit him to make his own selection. Individuals show a wide range of reaction to oxygen administration; patients in tents may exhibit wild symptoms of claustrophobia which are generally misinterpreted. Others may have a horror of face masks and the nasal catheter may cause nausea and wretching in certain cases. The wide use of tents in former years has given way to masks and catheters with the advent of air conditioning. It was long recognized by clinicians that the cool dry air within the tent was equally beneficial to the patient as well as the increased oxygen concentration. This was proved experimentally by Wolffe more than 10 years ago when he substituted compressed air instead of oxygen unknown to the patient. This is especially true in the hot humid days of summer. The need for tent therapy has thus lessened with air conditioning. The general body comfort of the patient has been found to enhance immeasurably the chances for prompt recovery.

Resistance to tent therapy because of claustrophobia focuses attention upon a reverse tendency in some patients who, after a period of such treatment, refuse to have the tent removed. A case in point is a 56-year-old lawyer who experienced a severe coronary closure with extensive heart muscle damage. For the first four days he was in marked shock and recovery seemed unlikely; he had received an impressive array of injections which he remembered. At the end of ten days he was well enough to be removed from the tent but he objected

a high solubility in blood plasma or equivalent electrolyte solutes may, however, be administered intravenously with little clinical reaction. Oxygen is readily soluble in saline and glucose solutions and the saturation of such solutions before injection may be useful. The actual amount of oxygen so administered is relatively small in comparison to the total overall oxygen requirements, but in desperately ill patients this may be the life saving margin. Direct injection of the gas into the venous circulation is not advised although there have been favorable reports on this method. Oxygen injection into the arterial tree is less objectional; in cases of marked cardiogenic shock due to massive myocardial infarction with drop in pulse pressure to 10 or 15 mm. Hg. or less, intra-arterial transfusion with artificially oxygenated blood has been successful. Oxygen thus administered is apparently rapidly absorbed by the tissues and embolism rarely occurs.

The usual route of oxygen administration is by inhalation. Experience has shown that there is little difference in oxygen utilization when given either by nasal catheter, face mask, or tent. Best results appear to be obtained with a saturation of 40% which is about twice the value of ambient air oxygen. There is now ample evidence from aviation and space medical research that oxygen concentrations greater than 40 to 50% do not serve any physiologic purpose; 80% to pure oxygen (100%) may cause pathologic and lethal changes when used over any long period of time. Most commercial gauges are adjusted to supply about a 40% oxygen saturation.

The choice of tent, mask, or nasal catheter depends

after breakfast. Every morning, he will tell you, he has an attack on his way to work; he can almost predict the exact spot where the episode will start. It may occur half way up or at the top of a given flight of stairs, or after walking an incline or hill to get to his automobile or other means of transportation. Many stop to see if the attack will disappear; if it does not or even becomes worse, nitroglycerin is used. This may become a familiar pattern in many patients' mode of living; it may go on for months or years. A statistical study of this group has shown that in about 8% the anginal syndrome may entirely disappear after a given period of time, but the vastly greater number eventually develops a frank coronary arterial closure, usually within 18 months. Some patients, however, may have an anginal history extending over 10 to 15 years; in one instance, a clergyman experienced frequent attacks for 22 years and finally succumbed to a staphylococcus pneumonia.

In many of these individuals, a nitroglycerin tablet taken a few minutes before the anticipated attack may entirely prevent its occurrence; in some, the painful symptoms are markedly reduced. This preventative or prophylactic use of the drug has not met with unanimous approval among a number of cardiologists; while admitting the temporary benefits they have pointed out that the warning signal of the syndrome is lost and that the hazard of a true coronary episode may be precipitated since the patient, free from pain, continues his activities. It has been emphasized before that the pathological demarcation between angina pectoris and acute coronary occlusion may be very small; the difference is

strenuously; removal for only a few minutes produced a long series of complaints. The situation remained unchanged for another two weeks at which time the plea was made that the tent was needed for another patient who had just arrived at the hospital. The lawyer insisted on buying a new tent for the patient so that he could keep the one he was using. He remained in the tent for a total period of 5 weeks before he could be persuaded to have it removed. Compressed air was attempted once but he spotted the tank which had a different color, and refused to have it set up. Similar reactions are often seen in patients with poliomyelitis who require respirators over a period of time; many object to removal from the tank even though the respiratory function has returned to a more or less normal state. It is a geneal rule that patients *should* be removed from an oxygen tent as soon as the indications for such therapy are no longer valid. Such indications are nausea, vomiting, hiccoughing, and restlessness; individuals in coma or in semi-conscious states are more easily treated in tents.

Prevention of Angina Pectoris

Prevention of the anginal episode has only received scientific research during the past decade; this is strange because patients for many years have learned from experience that nitroglycerin taken *before* an anticipated attack is even more salubrious than after the episode has developed. This is especially true in post-prandial angina pectoris; one of the most common histories is associated with the man who experiences his pain while walking

patient response to nitroglycerin after the development of the pain pattern, the preventative aspects of the drug may serve a very useful purpose and should be recommended.

The prevention of anginal attacks by the slower acting drugs has received favorable attention with the introduction of the various esters of the nitrate series. Of these, triethanolamine trinitrate biphosphate (Nitretamin, Squibb) and pentaerythritol tetranitrate (Peritrate, Warner-Chilicott) have a definitive action in certain patients. The development of delayed action drugs by differential solubility has produced a new group of remedies; Nitroglyn (Key) provides a continuous release of nitroglycerin over a 10 to 12 hour period from a 1/10 or 1/25 grain tablet. Coated granules of pentaerythritol tetranitrate (Corovas, Amfre-Grant) and Pentritol (Evron) permits a slow release of the active substances.

Aminophyllin has been in wide use for the past 30 years, over 60 different combinations are listed. Cardiologists differ in their opinion concerning the effectiveness of the drug in the prevention of anginal attacks. Repeated pharmacologic experiments have shown that aminophyllin produces a greater coronary flow in experimental animals and man when used in sufficiently large doses. These doses are from 5 to 10 times the average recommended amount for clinical use; in these amounts there is usually considerable gastrointestinal irritability. Patients complain of nausea, anorexia, gas, and occasionally vomiting and few individuals will continue to take the drug under these circumstances. Chiefly for this reason aminophyllin has been combined with other

the so-called clinical margin of safety. Insofar as the painful symptoms have a tendency to reduce or stop all physical activity, this may be the determining factor in the prevention of myocardial infarction. From this point of view, and there is much clinical evidence to the support the concept, the use of the drug to prevent an attack is not without some degree of risk.

On the other hand, proponents of the preventative use of nitroglycerin believe that the reverse physiologic reactions take place; if the postulate is true that the drug causes a rapid increase in blood flow throughout the coronary tree, then the heart muscle is better prepared for the additional work load required by patients' activities and the hypoxial factors ultimately responsible for the pain syndrome do not occur. The myocardium is thus spared from a temporary phase of reversible ischemia and while it is unknown how many such episodes may occur without permanent change, it is probably that each "attack" leaves an ill defined alteration in heart muscle physiology.

Recent experimental evidence seems to indicate that both concepts have some validity. Where there is considerable clinical data to suggest extensive and advanced coronary artery disease and its associated myocardial damage, the preventative use of nitroglycerin involves a calculated risk. Here, the margin of safety has been rendered low by pre-existing pathology and the extra work load permitted by masking the painful symptoms, may be sufficient to precipitate another occlusion. Conversely, where there is little or no objective evidence of coronary or myocardial disease and where there is a good

ful. Theominal (Wintrop), Theocalcin (Bilhuber-Knoll), Diurbital (Amfre-Grant), Thesodate (Brewer), Calpurate (Maltbie), Glytheonate (Patch) and Choledyl (Nepera). Elixophyllin (Sherman) is a liquid preparation which is useful in those patients who cannot take tablets for one reason or another.

Papaverine has long been favored by many cardiologists; it is the drug of choice in the prevention of anginal attacks. In certain individuals it may be habit forming and it may produce the symptoms of chronic narcotism when used over long periods. As with other drugs of the *opium series* like morphine, codeine, dilaudid, and pantopon, there may be a great variation in patient responsiveness. Subclinical dosage may produce a paradoxical effect in enhancing the symptoms instead of reducing them. In such instances the anginal sufferer may complain that he has more rather than fewer attacks when papaverine is first given and he may be reluctant to increase the dosage. Experience has shown that the drug can be started with 50 mg. and increased to 2 or 3 or even 4 tablets a day depending upon the patient's activities. Individuals on a papaverine regime should not smoke; recent studies have shown that smoking blocks the vasodilating effect of the drug both in the coronary and peripheral vascular tree. Smoking and angina pectoris will be discussed later in this chapter. The continued and uninterrupted use of papaverine for longer periods than 3 or 4 months is not advised; a reappraisal of the patient's general condition should be undertaken every 3 or 4 weeks. Some cardiac clinics employ the drug on an alternate week program, one week papaverine

substances in the attempt to eliminate or reduce the disagreeable by-effects. For example, aluminum hydroxide gel which is a familiar component of many gastrointestinal remedies, has been added to aminophyllin as in Aminodrox (Massengill) and Cardalin (Irwin Neisler). The barbiturate drugs have been combined with aminophyllin as in aminophyllin with pentobarbital (Searle) and with other dilator drugs as in Roniacol with Aminophyllin (Roche). These are but a few of the common preparations which are available.

A final word about aminophyllin; in spite of the difficulties of administration, it has proved to be a reliable substance in the treatment not only of angina pectoris but also in myocardial failure secondary to coronary disease, in certain phases of cor pulmonale, and in asthma with pulmonary edema. It is well tolerated in proper solution for intravenous medication (Dubin) and it is also valuable by suppository (Wyeth). It cannot be given subcutaneously or intramuscularly without severe local tissue reaction.

The theophyllin and theobromine group are better tolerated by many patients and in contrast to aminophyllin, may be given over long periods without patient rejection. Reisman once said that a drug is only useful if the patient takes it; an unused bottle in a medicine cabinet, according to Meyers, is a triple threat—to the patient, to the physician, and to the pharmacist. The theophyllin-theobromine drugs, while definitely less effective in the control of the anginal syndrome, frequently are of considerable value. The list of these compounds is a long one; in our experience the following have been very use-

and frequently is difficult to determine from the available clinical data, regardless of how thorough the physical and psychologic examination may have been. For this reason, the treatment of angina pectoris has over the past century been largely empirical and for this same reason a wide variety of drugs has been developed in the hope and expectation that one of the remedies may be specific in a given case. This is no confession of defeatism; it is an admission that many keys must be tried to open an unyielding door. The door here is the discovery of a drug or method which can be utilized to give relief to the patient. No apologies are necessary when the physician is forced to change medication repeatedly and I do not agree with certain authors who bewail the increasing list of new drugs offered by the pharmaceutical industry for the control of this disease. In its place and under given conditions each drug has a definite role.

Angina Pectoris and the Gastrointestinal Tract

While the anginal syndrome is primarily a disability of the cardiovascular system, the trigger mechanism may lie outside of the heart and blood vessels. The psychologic and psychosomatic implications have already been discussed, certain orthopedic problems like arthritis and injuries to the cervical and thoracic spine may precipitate anginal attacks. The endocrine system with the adrenals predominating have a definitive role in the trigger mechanism. The gastrointestinal system, however, has the largest part in the development of the chain reaction mechanism which eventuates in an anginal attack.

and the next week a substitute, perhaps of the theophyllin group. In most instances, papaverine reduces the need for nitroglycerin and the effectiveness of the drug may be estimated by the nitroglycerin count.

Paveril Phosphate (Lilly) is a synthetic non-narcotic substance which may be substituted occasionally for papaverine; it must be used in far greater dosage—0.5 to 0.75 gm. per day. Patients may complain of giddiness and tinnitus. Papavatrol (Kenwood) contains ethylpapaverine HCl and pentaerythritol tetranitrate; this is another non-narcotic substitute which is especially effective in the prevention of anginal attacks in elderly patients with hypertension.

Paracodin (Bilhuber-Knoll) is dihydrocodeine bitartrate; while more or less habit forming, may be useful in lessening the pain in very severe attacks of angina. In several instances this drug has been almost specific in the management of intractable heart pain.

The number of drugs employed for the prevention of anginal episodes is very large; the list continues to grow with each year. This is chiefly due to the fact that the initial trigger mechanism responsible for the development of the syndrome may result from many factors. The terminal stage of myocardial hypoxia secondary to inadequate blood flow is predicated upon many known and unknown physiologic reactions in which endocrine, humoral, biochemical, electrolyte, neurological, and mechanical mechanisms play a varying and competitive role. Insofar as anyone of these factors may predominate in any given case, effective therapy requires a careful evaluation of the basic pathology present. This may be

"Gas" is also a frequent complaint; distension of the gastrointestinal tract may be productive of many stimuli which can trigger an anginal episode. The two important areas appear to be in the stomach and transverse colon particularly at the splenic flexure. Gas in the stomach is usually the result of air swallowing; it is doubtful that fermentation occurs, except perhaps in conditions where there is a markedly delayed emptying time. Air swallowing while eating is a common habit in the personality types likely to develop angina; they tend to eat quickly and usually suck in air while drinking. Constant talking while eating is also a cause of air swallowing. Libby classifies the air swallowers into two types: the burpers or belchers and the non-burping or non-belching group. Those that can get rid of the gas are less likely to develop angina than those who do not. Patients frequently volunteer the information that heart pain is lessened or completely removed after gastric decompression by one method or another.

Accumulation of gas in the colon, on the other hand, is usually the result of intestinal fermentation; experiments have shown that air introduced in the stomach and which passes into the duodenal is usually absorbed in the first portion of the small intestine. Distention of the transverse colon is chiefly responsible for the high diaphragm syndrome which has been previously discussed; it should be repeated here, that elevation of the left leaf of the diaphragm particularly, is a common cause of substernal and precordial distress. Pressure on the heart and the structures within the mediastinum produces both severe and constant pain which may be lo-

The relation between gall bladder disease and angina pectoris has long been recognized and it may be said that the first surgical approach in the treatment of angina was surgery of the gall bladder and not surgery of the heart. Medical literature is replete with the successful management of heart pain as the result of such procedures and where there is indisputable evidence of such pathology the practitioner should seriously consider the relationship and advise appropriate surgical measures.

More common are the simple functional disturbances of the gastrointesinal system; these include the "heart burn" group. The very derivation of the concept of heart burn indicates the close relationship which has traditionally existed between the heart and stomach. Hyperacidity and angina pectoris are close companions in many anginal histories; both may be spawned by emotional and psychogenic stress. The candidate for ulcers and the candidate for angina pectoris have the same personality problems; the two conditions may occur together but more often one alone predominates. Weiss has stated with no little truth that such individuals face jeopardy at three levels; the brain, heart, and stomach. They may get a "nervous breakdown," angina pectoris or ulcers.

Some attention should therefore be given to the patient's gastrointestinal complaints; it is not uncommon to hear a patient state that the use of antacid remedies definitely decreases the incidence of his anginal attacks. The widely advertised alkalizers are in common use among many such patients; Tums, Alka-Selzer, Bromo-Selzer, Alkaids, Pepto-Bismol, are but a few which anginal sufferers carry with them in self medication.

was always characterized by nausea, vomiting, heart pain, and collapse; many so stricken died on the way to the hospital. All of these cases were probably acute coronary occlusions with myocardial infarction.

The relation of eating and physical effort in the development of the anginal syndrome is frequently noted in men who are forced by circumstances to hurry to work after a hasty breakfast. In a review of 600 cases of angina pectoris, made a few years ago, it was found that the condition first developed in the morning after breakfast while on the way to work in about 70% of the individuals questioned. In many instances, this was the only attack which the patient might experience during the entire day. It is possible that this post prandial effort was the most strenuous activity by the individuals; commuters who run after a train, motorists who are forced to open heavy garage doors or other preliminary chores before the automobile can be driven away, men who are required to climb hills or long flights of stairs to reach transportation facilities, those that carry heavy luggage, are but a few of the candidates who are likely to develop post prandial angina.

When such cases occur in the doctor's practice, the simple expedient of removing the gastrointestinal factors may be revealing, with all other circumstances remaining unchanged, the patient is instructed to eliminate his breakfast at home completely. When he arrives at or near his place of work, he may then eat; if the trip is a rather long one, there is no objection to eating on a train, for example, provided there will be no particular effort in

calized by the patient in the midsternal and upper chest area; pain may also be transmitted to the back of the chest and sometimes to the left shoulder. Such flatulence produced pain may be more or less relieved by expulsion of gas.

In some patients management of eating habits and diet may be the key to the successful treatment of their anginal attacks; experience has shown that most such individuals are more or less subconsciously aware of the close relationship of eating and heart pain. A word from the physician in regard to the *modus operandi* of the trigger mechanism may result in closer patient cooperation and prompt relief from the distressing and alarming symptoms of the anginal syndrome.

Post Prandial Angina Pectoris

The classical description of angina pectoris includes the picture of a well fed gentlemen leaving the warm comforts of his dining place and facing the cold wind on a winter's night. Physical effort after a heavy meal is frequently the cause of the individual's first anginal attack; it is also a common history in patients with acute coronary occlusion. It is interesting in this connection to point out that anginal sufferers rarely have nausea and they do not vomit; in acute coronary occlusion, nausea and vomiting are common and may occur even before the onset of pain. The familiar "attack of acute indigestion" which seized the after dinner speaker and which was well known to an older generation of practitioners,

TOBACCO

A special word about smoking and angina pectoris may be said here; most cardiologists believe that smoking has no place in any type of heart or blood vessel disease. The evidence against smoking in peripheral vascular disease is well established; smoking is definitely contraindicated in congestive failure. Diuresis may be hampered by smoking; the accepted concept is in relation to lessened renal vascular capacity by vasoconstriction. It is also doubtful that inhalation of tobacco smoke is beneficial in the lessened pulmonary function occurring in edema of the lungs regardless of its degree or its extent. Tobacco certainly has no place in hypertension, especially in systolic levels above 200 and diastolic above 110.

The tobacco question in angina pectoris is less conclusive. In instances where the patient volunteers the information that he has given up smoking because he has recognized that tobacco has increased the number or severity of the attacks, there can be no doubt that smoking is definitely contraindicated. However, many patients will state that moderate smoking seems to have a soothing effect which may ameliorate some of the tensions which subsequently could lead to an attack. This brings up the perennial problem which every practitioner must face concerning the definition of "moderate" smoking.

Research by the tobacco industry has clarified a number of questions which have clinical implications; the chain-smoker, for example, is now recognized as a spe-

getting from the railroad or bus station to his work. In most patients, the morning attack may be completely removed or it may occur in a very mild form. Nitroglycerin, if routine before, may not now be needed.

The amount of food consumed at breakfast may have little relation to post prandial angina; the standard American breakfast includes orange juice and coffee plus the traditional special items. A cup of coffee in certain patients may be just as productive of the post prandial syndrome as a full meal. If the no breakfast experiment results in cessation of the anginal attacks, a gradual increase in small amounts of food may be permitted up to the point when the episodes tend to recur again. Given an intelligent and cooperative patient, many morning attacks may be removed by this simple arrangement of eating habits.

Patients with angina pectoris should be instructed that all types of physical activity will be better performed on an "empty stomach"; in athletes the best performance is always attained before eating, never after. All animals, including race horses whose diet is very carefully controlled, work better when gastrointestinal activity is at its lowest ebb. If an individual knows that he will be required to expend physical energy for a necessary purpose during his working day, eating should be postponed until such demands have been completed. Many anginal sufferers are fully aware of these facts but unless their attention is forcibly directed to cause and effects they may continue to experience episodes, a word from the physician may eventually be life saving to the patient.

ivity; all other matters are usually spoken openly and reely with all concerned but the question of sex may preoccupy the anginal patient above all other considerations and he usually wishes to speak to the doctor alone. The question is a straightforward one and deserves an adequate and competent answer in every case. An unresponsive attitude on the part of the physician or any intent to make light of a situation which may loom high in disturbing the patient's peace of mind may be responsible for a number of untoward conditions.

Certain facts are well recognized; if the physical activity of intercourse produces an anginal attack, the answer is of course simplified. Any activity which causes an attack is contraindicated. On the other hand, all of the preventative measures used in suppressing the syndrome may be employed here with equally satisfactory results. First, intercourse should not be performed after a meal; post prandial sexual activities have been responsible for many anginal attacks as well as acute coronary occlusion. Secondly, the preventative use of nitroglycerin may be very effective here; and thirdly, most of the physical activity should be transferred to the partner in the act.

Another important factor is the previous sexual life of the patient, some men after a given age which varies from patient to patient may experience a more or less complete loss of interest. The chief problem here is in regard to their sexual companion; if complete indifference occurs in both parties, the answer has few or no complications. Where, however, the wife retains an interest in such normal companionship the patient may feel

cific personality type characterized by anxiety neurosis, tendency to have ulcers, hypertension and cardiovascular degenerative diseases. A chain-smoker is an individual who consumes 40 to 60 cigarettes a day or more or 6 to 10 cigars. There do not appear to be many excessive pipe smokers. Chain smokers will not stop voluntarily until the basic psychogenic problems have been solved. But it is interesting to note, that these patients when forced to stop smoking by an attack of coronary occlusion or any other serious condition, lose the habit without any special change in the psychogenic factors.

Tobacco is used for many reasons, many of which are fancied and some of which have a very practical background. Insofar as smoking in certain individuals may exert a soothing or quieting effect which in many respects resembles the action of the tranquilizers, it may have a very useful purpose in subjects who require support during psychogenic stress. In the chain reaction mechanism, already described in the development of the anginal syndrome, it is quite possible that such a barrier effect may carry a susceptible or responsive patient over the stimulus period. The selection of such patients requires good judgment on the part of the physician; he must balance the good and evil effects of the common tranquilizers with similar good and evil effects of tobacco in each individual case.

Sexual Activity

The chief subject discussed behind closed doors in every practitioner's office is the question of sexual ac-

as a specific endocrine benefit to be expected from coitus. While this is a greatly oversimplified statement in regard to the relationship of the sex steroids and heart disease of the degenerative types, it may serve as a basis for discussion in regard to the question which is often raised by patients and their wives concerning the need of sexual activity.

If the patient states that he feels generally and constitutionally better as a result of intercourse without being able to precisely define what he means by "better," it is likely that he has received the assumed benefits of steroid activity. Many patients volunteer this information readily; others either have no special reaction to the secondary effects of coitus or they may feel depressed and their condition may worsen the few days following the act. In the latter group, intercourse should be limited.

A final word about sexual activity after a coronary occlusion, the same problems which arise in angina pectoris also occur in answering the question of when coitus can be safely resumed after a myocardial infarction. Ordinarily, no pain factor is involved in post coronary patients; dyspnea and palpitation may occur and acute pulmonary edema has developed in some patients. What are the tests of safety? From a scientific and practical standpoint, the tests for functional capacity of the cardiovascular system should provide the physician with sufficient factual data to provide a satisfactory answer in the individual case, it has been estimated that energy expenditure in normal coitus is equal to about 5000 foot/pounds of work performance. If the subject is able to score an average Cardio-Pulmonary Index, for example,

obligated for reasons of personal pride and ego satisfaction, to perform his marital duties in spite of any disability which he may suffer. Here, a sympathetic talk with the wife and an explanation of the alternate possibilities should be undertaken by the physician.

Men with previously active sexual lives may be loth to discontinue a more or less important facet in their existence. They frequently bear the pain of an anginal episode with understanding fortitude; the hazard here is in relation to the possibility of coronary occlusion. The sexual drive in such individuals frequently exceeds normal prudence. Here, caution must be exercised and a program of preventative anginal measures must be strictly adhered to.

Is sexual activity necessary? The trend toward the use of the androgenic and estrogenic substances in angina pectoris and coronary artery disease is based chiefly upon the experimental work, correlated with the development of the atheromatous syndrome in animals, by Wolffe, Katz, Jablons, Hueper, Steiner, Duff, and many other investigators. There is considerable clinical evidence to show that the sex steroids have a specific effect in cholesterol metabolism and insofar as disturbances in cholesterol chemistry are responsible for the degenerative changes which lead to angina pectoris and coronary thrombosis, the use of the andro-estrogenic substances may have a definitive place in the treatment of these conditions. Pomeranz postulated that during the sexual act, increased amounts of androgenic substances as well as other associated steroids were released for essential metabolic balance and that all other conditions being equal, there

and under the circumstances the practitioner should consider the syndrome to be a coronary episode and treat the patient accordingly. It has been mentioned before that such patients may require a week to 10 days observation; if the clinical and laboratory studies remain more or less unchanged, the attack was probably angina pectoris although doubt may subsequently be cast on the presumptive diagnosis.

Cardiogenic shock is always a dramatic and alarming symptom in coronary occlusion; the type and degree of of the shock may bear no special relationship to the extent of the actual pathology present. Autopsy findings in patients dying in extreme shock may show minimal changes whereas other individuals without shock who have died suddenly from ventricular fibrillation or other types of real or functional cardiac arrest frequently show massive areas of myocardial damage. There have been no consistent correlation findings between the area of infarction and the development of shock. Shock may develop rather promptly after the onset of pain; usually it occurs after a few hours. It may be delayed for 24 hours or longer, it is possible that when shock is so delayed that another occlusion has taken place. Multiple occlusions which have occurred several hours or days apart are not an infrequent post mortem discovery.

Shock may develop during or directly after the nausea and vomiting phase of the attack; the pale facies and the cold clammy sweating of the *head*, *neck*, and upper chest, usually associated with retching regardless of the cause, may merge with similar symptoms due to cardiogenic shock. More often there is an interval of minutes

the physical activity of intercourse is not to be feared; if, on the other hand, the post exercise electrocardiogram shows significant changes suggesting coronary insufficiency, caution is advisable. The post exercise Respiratory Index is also a simple guide for the examiner to utilize.

In summary, these are some of the problems which the practitioner must face in regard to the sexual life of the patient; their importance will vary from case to case. In some instances, the question will be emphasized out of all proportion when compared to the interest concerned in the individual's other, more important aspects of his disability. It may be the patient's sole disturbing factor of his disease. In others, the question may play a minor role in the management of the condition; with them there is no problem.

Cardiogenic Shock

Patients with angina pectoris rarely go into collapse; if shock or prostration occurs, the episode is more likely to be due to coronary thrombosis with occlusion. However, the pain of an anginal attack may be so severe that the individual appears to be *in extremis*; subsequent electrocardiographic studies in such instances may reveal no changes, although, some cardiologists believe that all such attacks are probably associated with subminimal myocardial involvement. On the other hand, certain coronary occlusions ushered in with severe pain may likewise show no ECG alterations. The differential diagnosis between the two conditions may not be possible

monary edema is present it may be even difficult to identify the sounds. Murmurs frequently develop during shock; these murmurs are nearly always systolic in time and are heard best at the apex. They may be louder than the heart sounds and may change their characteristics many times during a 24 hour period; they are functional in origin and probably due to relaxation of the mitral ring with its accompanying functional insufficiency. In certain cases, functional systolic murmurs of the tricuspid valve also occur; this is seen not infrequently with pulmonary edema. The loss of the heart sound pattern and the development of murmurs during cardiogenic shock does not necessarily worsen the prognosis; it is a part of the shock syndrome and usually disappears with better restoration of ventricular output.

Arrhythmias in Shock

Arrhythmias are not uncommon in shock from coronary occlusion; premature beats may occur promptly. Paroxysmal atrial fibrillation with and without flutter also is noted in many cases; more serious are the ventricular paroxysmal tachycardias which first occur in short runs lasting but a minute or two. Later, the rhythm may persist for several minutes or hours; the danger here is the transition to ventricular fibrillation with sudden death from functional cardiac arrest. The conduction disturbances may also occur but these are less common; as a group, the P-R interval leads the list, with interventricular block patterns and complete heart block with atrial and ventricular dissociation following in frequency. In general,

or perhaps hours before the shock phenomena develop and continued nausea and vomiting may become one of the most troublesome and serious complications in the management of the case.

Blood Pressure in Shock

Drop in blood pressure occurs in all types of shock; in cardiogenic shock it constitutes one of the most alarming symptoms. Neither absolute systolic or diastolic levels have as much significance as pulse pressure; hypoxial phenomena develop when pulse pressure is less than 15 to 20 mm. Hg. A fall from 180/90 to 100/60 will ordinarily produce fewer symptoms than 120/80 to 100/80; in shock, 80/70 may be more serious than 60/30. Diminution of pulse pressure is associated with a feeble or difficult pulse to palpate; experiments show that the pulsations of radial artery tend to disappear when pulse pressure falls below 20 mm. Hg., regardless of the systolic levels.

Heart Sounds and Pulse Rate

There may be no correlationship between poor and impalpable pulse and the intensity of the heart sounds; usually the first heart sound syndrome is noted. As previously described, this change in the normal heart sound pattern occurs most frequently in left ventricular failure; only one heart sound can be heard and this is the first which may be accentuated above its usual intensity. Distant and poor heart sounds are common; when pul-

cal factors in respiration. The high diaphragm syndrome resulting from gastric and transverse colon distention will also render respiratory function more difficult. Insofar as morphine in certain responsive individuals may lessen gastrointestinal activity, respiratory movements may be embarrassed. Occasionally, it is almost impossible to control the pain factor; the patient's restlessness and threshing around may add a considerable load to an already overburdened cardiovascular system. The use of a general anesthetic may be employed; ether given by the open method is the procedure of choice. Ether also increases oxygen partial pressure of the arterial blood and may serve to ameliorate some of the hypoxial symptoms. Ether has also been used in the water bottle hydrating devices employed in oxygen administration; a 10% mixture of ether and water may render oxygen therapy more effective in pulmonary edema. A word of caution must be noted here, oxygen-ether mixtures are highly *explosive*. All electric contacts must be carefully grounded and no open flames, such as from alcohol lamps, permitted in the room which should, of course, be well ventilated. It should not be necessary to speak of the NO SMOKING regulations.

Pulse Pressure Crises

When pulse pressure drops to critical levels in cardiogenic shock, specific treatment is promptly indicated. A number of drugs have proved to be effective; the chief of these is Norepinephrine or Levophed Bitartrate (Winthrop). The substance is given intravenously, about 4

the development of an arrhythmia has clinical significance chiefly in relation to ventricular output; it is usually of the least importance in premature beats and most in the ventricular tachycardias. Complete cardiac arrest from sinus nodal pacemaker block responds more quickly to resuscitation measures than functional cardiac arrest from ventricular fibrillation; in nodal block, the whole heart ceases functioning and no blood leaves the left ventricle, while in fibrillation of the ventricles, the atria are still contracting but there is no ventricular pressure gradient rise sufficiently great to open the aortic valve even when the diastolic pressure is zero or only a few millimeters above. There is no actual standstill of the heart here; in fact, the heart usually shows wild movement in contrast to the complete inactivity seen in pacemaker failure. In both instances, however, the end result is no ventricular output and collapse of the circulatory system occurs.

Management of Pain

With the development of shock the pain factor usually diminishes; some patients may go into shock without pain. Control of pain in the beginning of the episode may lighten the time and extent of the shock phenomena; it is doubtful that it will prevent shock from occurring. The use of the narcotic drugs has already been described; morphine is the least desirable of the group because of its secondary vagal effects. The chief of these is respiratory depression which may result from both central and local effects; since most men are diaphragmatic breathers, any phrenic-vagal dysfunction will enhance the mechani-

to occur. Parenteral medication is required; gastro-intestinal functional absorption ceases during shock. The former question concerning the use of the digitalis drugs in the acute coronary syndrome is now chiefly academic; the belief that the drug had a tendency to increase the irritability factors of the myocardium which might lead to the development of arrhythmias was based, originally, upon experimental animal work; most cardiologists believe that the calculated risk of impending heart failure and complete breakdown of cardiac function is greater than the possibility of drug induced irregularities. Moreover, when spontaneous supraventricular disturbances of rhythm occur, the drug must be administered, especially if the rate is rapid. Digitalis must be given cautiously in cardiogenic shock. The calculated dose is given directly into the intravenous tubing which is carrying the Levophed glucose solution rather than placed in the bottle containing the mixture. Experience has shown that the greatly diluted concentration of the drug is much less effective and less likely to produce the anticipated results. A number of digitalis preparations are available and all may be used satisfactorily; the physician should administer the product with which he is most familiar.

Digitaline Nativelle (Varick), Crystolgin (Lilly), Digitoxin (Abbot), Digifolin (Ciba), Digiglusin (Lilly), Gitaligin (White), Lanoxin (Burroughs Wellcome), Cedilanid (Sandoz), are but a few of the digitalis glycoside fractions which may be suggested. The strophanthin drugs have found greater use in Europe, while these products have a more immediate action, the margin of clinical

mg. per 1000 ml. 5 to 10% glucose solution; the rate of injection must be carefully controlled using pulse pressure as a guide. Optimum results occur when pulse pressure is increased to 25 to 30 mm. Hg.; the actual systolic level is less important. Infusion should be continued as long as necessary; after 24 hours there may be considerable local skin and tissue reaction unless extreme precautionary measures are observed in the injection technique. One injection site is better than several; other suitable veins should be reserved for additional necessary therapeutic injections. In extremely sick individuals many other drugs may have to be used intravenously; a familiar but disconcerting hospital patient is one whose "veins are all used up." This should be avoided wherever possible and the Levophed injection should be regarded as a surgical procedure and not just "another intravenous." Memphentermine sulphate or Wyamine (Wyeth), Methoxamine Hydrochloride or Vasoxyl (Burroughs Wellcome), and Phenylephrine or Neosynephrin (Winthrop), may also be employed; these latter drugs are also useful in suppressing a number of the arrhythmias which may develop in cardiogenic shock and the selection of a given substance should take its side effects into consideration.

Congestive Failure in Shock

When congestive failure develops early in shock, one of the intravenous digitalis glucosides should be promptly administered. The early discovery of basal rales is sufficient indication for digitalization; it is not necessary to wait for a reflex cough or for widely scattered crepitation

will accomplish the same change in hemodynamics. The Jarcho bed with its inclined plane mechanism and the Henry pivoted bed permits exact adjustment of the desired position. In emergency situations, where the patient is in profound shock, the Cramp position may be useful; the individual is placed on the floor with his head resting on a pillow and his body and legs are held high on the edge of the bed or chair. Here, again, the calculated risk in increasing pulmonary edema must be weighed against a better blood supply to the brain; patients in this position must be watched and the position changed after 15 or 20 minutes. Most of these positions may be utilized within a tent but the face mask or catheter method should also be available.

The concept of position in cardiogenic shock is based upon the physiologic postulate that in such an emergency life depends upon an adequate circulation of the brain and heart. As volume output decreases, blood tends to be pooled in the extremities and abdominal viscera. Temporary separation of the arms and legs from the circulation may be accomplished by tourniquets; if 4 blood pressure instruments are available a variable type of arterial occlusion is possible. Using the right arm as a control, it may be noted that a rise in blood pressure occurs spontaneously when the two legs and other arm are thus shut off from the vascular tree. The rise may be as much as *20 to 40* mm. Hg. and insofar as this improvement in cardiac and cerebral blood supply may spell the difference between a rapidly impending fatality and the securing of additional time for other measures, the procedure has much to recommend it.

safety is much less. Ouabaine Arnaud (Varick), Strophantin (Burroughs Wellcome), and Unitroxin (Tilden), may be used in selected cases. Where pain and congestive failure occur simultaneously, Groedel suggested large doses of strophanthin; he postulated that if the pain was due to the relative hypoxia accompanying the failing myocardium, improvement in the contracting function of the heart muscle would increase volume output and hence produce a better coronary blood supply. However, this therapeutic chain reaction is also the goal of the *digitalis drugs and under carefully controlled conditions* these may be used with less danger.

The Orthostatic Factors

The use of oxygen has already been presented; in cardiogenic shock it may be the first line of therapeutic defense. A word about the position of the patient should be made; in pulmonary edema, orthopnea, and in other conditions where the individual has difficulty with respiration, it has been pointed out that elevation of the head and chest reduces the orthostatic factors and increases the comfort of the patient. In shock, there is usually marked cerebral ischemia which is proportional to fall in blood pressure and height of the head above the level of the aortic valves; here, priority must be given to increasing blood flow to the brain at the risk of increasing breathing problems. The patient's body should be placed head and chest down with pelvis and legs elevated; the adjustable hospital bed simplifies the procedure but simple raising of a household bed at the foot

upon the time factor of the two procedures. When the injection is given first, the brain and heart benefit. The venesection is then performed and the pulmonary bed is relieved of its burden. The method is not recommended in general shock treatment but in selected cases where previous drug therapy has failed, it has been successful as a measure of last resort and may be borne in mind.

Anticoagulant Therapy

The use of the anticoagulant drugs in cardiogenic shock as well as in the subsequent management of coronary thrombosis with myocardial infarction deserves a special section for discussion. This is due to many considerations for it is doubtful whether any method of treatment in recent medical history has received as much acrimonious debate as the anticoagulants. Two violently opposed schools of thought still publish lengthy statistical data for and against its administration in heart disease. For the practitioner, a middle ground dictated by experience and caution, tempered by expediency, is recommended. In this discussion the author cannot remain neutral but must present the facts as he has seen them since the introduction of the method over 10 years ago.

At the very beginning it must be pointed out that a strange change has occurred in the focus of attention between the two schools, debate centers not upon the question of when to *start* anticoagulant therapy but when to *stop*. If the drug is to be given it should be started as soon as the diagnosis of *myocardial* infarction has been made, the indications for its use are still ill defined and

Pulmonary Edema in Shock

Acute pulmonary edema in shock should be handled in much the same manner as when this syndrome appears in other conditions; it is likely that the digitalis preparations have already been administered; the aminophyllin drugs should be given promptly by intravenous injection but not at the same side as Levophed or other similar substances. It is better employed in concentrated form and should not be mixed with large volumes of other solutions which may be in the process of intravenous administration.

Phlebotomy in shock poses a number of problems; in acute pulmonary edema, as for example, in mitral stenosis, it has been shown that venesection may be life saving. The symptomatic improvement of the patient is prompt and clinical changes can be noted quickly; here the overloaded right heart is released from retrograde pressure factors and restoration of a more normal circulation occurs. In cardiogenic shock, there may actually be an increase in circulating fluid but it is pooled in non vital areas; intravenous injection, while adding to a peripherally burdened circulation, is an attempt to supply the vital areas with an increased volume/pressure gradient. This anticipated favorable action increases the pulmonary vascular load and enhances the factors responsible for pulmonary edema. The paradoxical situation of injecting glucose solution into the vein of one arm and removing an equal amount of blood from the other, is not without certain important clinical implications which depend

bot) is perhaps the best known. Others are ethyl biscoumacetate or Tromexan (Geigy), phenidione or Hedulin (Walker), diphenadione or Dipaxin (Upjohn), phenprocoumon or Marcumar (Hoffman-LaRoche), acenocoumann or Sintrom (Geigy), and cyclocumarol or Cumopyran. The warfarin sodium group are represented by Coumadin (Endo) and Prothromadin (Harvey).

The therapeutic fixation of prothrombin time at twice that of normal is the goal of anticoagulant management; during the first week or ten days, this may require a certain amount of "dosage juggling" since the required amount of the drug can only be determined by trial and error. Fortunately, overdosage is readily controlled by the vitamin K substances, here, Adrestat (Organon), Mephyton (Merck, Sharp and Dohme), and Synkayvite (Roche) produce prompt results, returning prothrombin time to within normal limits in minutes.

Absorbic acid should be administered to all patients on anticoagulant therapy; it has been estimated that 5 to 10 times the normal vitamin C daily requirements are essential for capillary integrity. The tendency to capillary fragility is lessened but not entirely eliminated, the threat of hemorrhage starts with changes in the capillary mechanism. Attempts to correct this tendency with the rutin drugs have led to complications; rutin may neutralize the therapeutic effects of the anticoagulants and is theoretically contraindicated. However, in certain selected cases where capillary fragility is noted by small ecchymotic spots on the skin and where such purpuric areas develop even when the prothrombin time has returned to within normal limits after a test withdrawal period of the anti-

depend chiefly upon the experience and belief of the physician. Protracted pain, the onset of congestive failure, evidence of pulmonary infarction or peripheral embolism, chronic deep thrombophlebitis of the legs, patients debilitated from previous long-standing diseases or bedridden from surgical or orthopedic conditions, and certain individuals who are restless and agitated are some of the most important compelling reasons. Yet, patients with none of these symptoms are just as likely to develop embolic complications; it is frankly admitted, however, that the incidence is lower than the first group in nearly all statistical studies, but in any given case, the possibility is always present.

Simultaneous heparinization is suggested when anticoagulant therapy is started; Depo Heparin Sodium (Upjohn), Heparin Repository (Philadelphia), Heparin Sodium (Lilly), Liquaemin Sodium (Organon), have proven to be satisfactory. Hep-Nine B Fortis (Columbus) contains Heparin Sodium, Choline Chloride and Vitamin B_{12}, a combination which has a clearing action in the so-called giant cholesterol molecules seen in advanced atherosclerotic disease with involvement not only of the coronary arteries but also of the liver and kidney. The heparin effect is usually produced promptly and therapeutic results occur within a few hours.

The hydroxycoumarin drugs require a much longer period to reach the desired blood levels; the usual large initial dose shows a prothrombin reaction in 18 to 24 hours. Occasionally, full effect is not obtained for 48 hours. A number of reliable anticoagulant substances are now available; bishydroxycoumarin or Dicumarol (Ab-

the remaining third constitutes a group where the practitioner must weigh a number of immediate and remote factors before coming to a considered decision.

Some of the immediate problems involve the question of expediency; the anticoagulants demand accurate laboratory control. Prothrombin time determination, while not the most complicated laboratory procedure, is also not the simplest. The prothrombin time must be made once or twice a day for the first 72 hours, then every day for ten days to two weeks. The schedule thereafter may include a test every other day with lengthening of the intervening period to twice a week, and then once a week for several months. At some point in the after care, the test may be carried out twice a month and in certain cases only once a month. Unless the physician has such laboratory facilities available, the drug is used without effect in too small doses and with hazardous results in too large. Until simpler methods of estimating prothrombin time are developed and may be employed by the practitioner himself, anticoagulant therapy will necessarily be limited within the frame work of technical control.

coagulants, rutin in combination with vitamin K_1 should be given; Rutorbin (Squibb), Rutascorb (U. S. Vitamin), Rutin and Ascorbic Acid (Sherman) and (Upjohn), and Rudionate-C (Ascher) are suggested.

Not all patients require or should be given anticoagulant therapy; the author believes that many individuals run an uneventful course during a coronary episode. It has been pointed out previously that in 20% to 35% of the individuals stricken with an initial attack, most symptoms tend to lessen or completely disappear within 72 hours. Both the electrocardiograms and laboratory data may show evidences of myocardial infarcation but the patient may be entirely symptom-free. In others, the objective evidences of the condition may be limited to a few positive observations. Many of these cases make a complete recovery and have no further episodes.

On the other hand, patients in shock, with congestive failure, and with evidence of embolization, are definite candidates for anticoagulant therapy regardless of all other considerations. Patients who have had a previous coronary closure and myocardial infarction regardless of the severity of the present attack should receive the drug. Patients who have a history of atrial fibrillation and flutter or recent episodes of paroxysmal tachycardia where the possibility of embolization is enhanced by the additional coronary-myocardial pathology, may be classified within the group where anticoagulant treatment is indicated.

In short, it has been demonstrated that in nearly all large series of cases of acute coronary occlusion which have been studied, about one-third show definite indications for anticoagulant therapy, one-third do not, and

tachycardias; individuals may exhibit similar hypoxial symptoms at both extremes of ventricular activity. Ventricular tachycardias at 280 may have the same functional output as complete heart block with an idioventricular rate of 10; both may show the same cerebral, pulmonary, and peripheral vascular complications of lessened blood supply.

Physical examination of such patients requires careful correlation of apical and radial rates; blood pressure apparatus is important not so much for the determination of systolic and diastolic levels but for a study of changes in ventricular output. Given a regular rhythm, variations in the systolic level from beat to beat, may have diagnostic implications. Graphic methods are frequently necessary in both slow and rapid irregularities in order to identify the specific arrhythmia; the electrocardiograph is the most important instrument here but the arteriogram, ballistocardiogram, and phonocardiogram may also reveal significant data.

Extrasystolic Arrhythmias

The general characteristics of the premature beats have been previously discussed; the correlation factors of apical and radial rates in this common irregularity have been emphasized and the significance of effective and noneffective beats has been pointed out. Attention has also been directed to the T-wave changes seen in the next normal beat following an extrasystole in coronary insufficiency.

The skip or jump in the regular sinus rhythm may be

CHAPTER X

THE ARRHYTHMIAS

Sudden change in pulse rate or rhythm may be productive of many subjective symptoms; heart consciousness, pain, dyspnea, weakness, and syncope may occur. There is no correlation between the type of arrhythmia and such symptoms nor in the severity of the irregularity and the patient's complaints. Responsive individuals may suffer just as much disability with simple premature beats as with serious types of paroxysmal ventricular tachycardia; likewise, atrial fibrillation and other irregularities may be unnoticed by the patient.

The history of the onset of the arrhythmia, if obtainable, may offer a number of diagnostic criteria; those that develop very suddenly may be distinguished from the group which have an apparent slow beginning or which have been present over a long period. The paroxysmal tachycardias, as the name implies, have an abrupt beginning and usually cease just as abruptly. They are divided into two general groups; those with regular or nearly regular rapid rates and those with grossly irregular rapid rates. Rapid rate may be defined as 180 and above.

Very slow (40 and less) rates both regular and irregular may also produce nearly all the complaints seen in the

lation is postulated on the action current of injury phenomenon in the infarcted areas of the heart muscle.

Ectopic foci may arise in many parts of the heart; the most common areas are at or near the sinus nodal pace maker, in both atria, in the junctional node area, in both ventricles and less frequently in the septal areas in or near the bundle system. The supraventricular ectopic rhythms are divided for clinical purposes into two types; occasional extrasystoles and premature beats in long sequences. The simple premature atrial beat, unless it interferes materially with ventricular output, ordinarily requires no treatment except, perhaps, medication tending to suppress the patient's subjective sensations or anxiety. Here, the tranquilizers may be used very successfully; indeed, the various chemical combinations of meprobamate have largely replaced the former use of the barbiturates in the symptomatic management of several cardiac irregularities as well as the premature beat group. When ectopic atrial beats dominate the rhythm of the heart, they may resemble or be identical with paroxysmal atrial flutter especially when the rates are 250 to 300; here, the vagal pressure procedures may be successful in culminating the attack.

Vagal Stimulation in Paroxysmal Tachycardia

A word about vagal pressure in the paroxysmal tachycardias should be made. The vagal reflex may be secured from a number of sensitive pressure areas; the carotid sinus offers the most consistent and effective pressure point. Pressure on the eyeballs, stimulation of the gag-

productive of many reactions in responsive subjects; it is the most common disturbance for which patients seek medical advice and it is the one irregularity which does not yield to the examiner's tendency to make light of the situation even though he may explain its relative insignificance. Formerly thought to be chiefly due to psychogenic factors, recent studies have shown that a large group of physiologic mechanisms are involved in the development of an ectopic focus sufficiently effective to interrupt the normal electrodynamic cycle. Such a focus must initiate an electric difference of potential which is greater in magnitude than the repolarization gradient in both atrium and ventricle. Such changes involve electrolyte *reversal activity of the cell membrane and represent* a so-called build up of the neutralizing potential to the point when it escapes from the inhibiting electric field. The factors favoring ectopic beats are interruption of blood supply, lateness in the diastolic period, and a slow sinus rate. The time factor can be determined by the simple test of exercise; most premature beats disappear when rate increases. Ectopic beats which persist at rates of 110 and over are more serious and are probably due to irreversible alterations in blood supply to the myocardium. Premature beats which arise in myocardial infarction represent the most significant type of ectopic initiatory stimulus; the difference of electric potential displayed by a dying heart muscle segment may reach very high amplitude approaching the range of the action current of injury exhibited by other types of muscle. The concept of functional cardiac arrest secondary to the rapid release of such ectopic stimuli in paroxysmal ventricular fibril-

breath, or speeding up respiration, may cause the ectopic rhythm to disappear; exercise may produce the same effect. There may be a tendency for the premature beats to recur again, but if the patient is instructed in the relationship, many anxious moments in his existence may be eliminated by this simple procedure which he can practice at will.

There is no specific medication for premature beats; attention to the patient's gastrointestinal problems will aid in many cases. The use of the tranquilizers has been mentioned; Miltown (Wallace), Compazine and Thorazine (Smith, Kline & French) and others of this group are worthy of trial. There is a long list of phenobarbital combinations which have been used with more or less success. Digitalis should not be used; it may enhance the forcible contractions of the extrasystole and may also increase the frequency of the ectopic rhythm. Smoking by an indirect mechanism may produce premature beats; some patients volunteer the information that such beats are more likely to occur when they are using tobacco. If cause and effect are thus established, the patient's habits require supervision, this holds true for certain foods and drinks. Those which have a tendency to produce gas may have a role in the development of ectopic beats.

Atrial Fibrillation

Atrial fibrillation may occur in paroxysms lasting but a few moments but sometimes for hours or days, it may develop in both normal individuals and in patients with

ging reflex, and in certain cases, stretching of the rectal sphincter, may be sufficient to stop an attack. A few seconds pressure, repeated if necessary several times, is usually more effective than long continuous pressure; the reflex mechanism tends to weaken on long stimulation but its rapid recovery will permit maximum stimulation again after 10 to 15 seconds resting period. Pressure on the eyeballs requires certain precautions; temporary and permanent damage of the structures of the eye, and particularly to the retina have occurred in the overenthusiastic endeavors of the interne staff to stop attacks in the hospital emergency room where these cases are most commonly seen.

Ventricular Premature Beats

Ventricular premature beats are frequent in patients both with and without heart disease; they are commonly associated with organic and functional disturbances of the gastrointestinal tract. Pressure exerted on the apex of the heart during the high diaphragm syndrome may be responsible for the condition; such pressure may cause changes in the superficial coronary circulation at the compression area, sufficient to produce the factors previously described in the mechanism of ectopic beats. Extrasystoles which occur after every third, fourth, or fifth normal beat, may be correlated with diaphragmatic movement. Thus a respiratory rate of 18 with a pulse rate of 72 might show a regular pattern of a premature beat in relation to every fourth normal beat. Holding the

mal hearts, there may be a moderate ventricular response to the increased demand for a greater volume output; it is postulated, here, that the sinus nodal pacemaker is still functioning and speeds up on normal accelerator stimulus. If the normal excitatory stimulus is greater than the ectopic mechanism responsible for the atrial fibrillation, these extra stimuli reach and pass through the junctional area with a resulting ventricular contraction. The original Winterberg test of functional integrity of the sinus node in atrial fibrillation was based upon this observation; in actual clinical application, if there is an appreciable increase in the apical rate after exercise, the pacemaker is probably operating and the case is suitable for conversion back to sinus rhythm. Where, on the other hand, there is no increase in the ventricular rate, conversion is not likely to be desirable. The tests still has certain implications which will be considered later in regard to conversion of rhythm.

The rapid types of paroxysmal atrial fibrillation do not respond to vagal stimulation and exercise produces minimal effects. The attack may subside with rest, sedation, and lessening of the anxiety factors which may be the predominant part of the syndrome. The danger of congestive failure with various grades of pulmonary edema are present if the episode persists longer than several hours. Digitalis and quinidine are the most effective drugs in the management of the condition. Since quinidine may induce certain sensitivity problems, digitalis is preferable unless the patient's favorable reaction to quinidine is known from previous experience.

widely different types of heart disease. The onset is usually abrupt and the rate may be very rapid; in contrast to certain other types of paroxysmal tachycardia, the rhythm is grossly irregular and a marked pulse deficit occurs. It is not unusual to note an apical rate of 180 and a radial of 100; counting the radial pulse is more difficult than determining the apical beat.

Paroxysmal atrial fibrillation may also occur with relatively slow rates; when the rates are from 56 to 88 per minute there may be no pulse deficit. In such instances the arrhythmias may sometimes be difficult to diagnose without instrumental examination. Generally, the patient is totally unaware of the change in rhythm but other individuals report that an ill defined "something had happened to them" without being able to precisely describe what is wrong. The slow "fibrillators" may experience a number of such episodes over a period of months or years without noting any other cardiovascular disability. Volume output is always diminished in atrial fibrillation but in subjects with slow rates, who do not perform more than routine physical effort, the loss is minimal compared to that of normal sinus rhythm. The drop in volume output is only noted when unusual work is required; like the hemodynamic phenomena occurring in complete heart block, the accelerator stimulus which normally speeds the pacemaker mechanism is functionally absent and there is no change in ventricular rate to meet the demand for increased blood supply. Symptoms of hypoxia may develop rather quickly with dyspnea, precordial pain, and cerebral vascular complaints of dizziness and syncope predominating. In slow fibrillators with more or less nor-

thrombi may develop in a few minutes in certain conditions, involving increase in the stickiness factor of the blood cells, loss of the wetness factor of the endothelium, and increase of the coagulating factors of the plasma. The formula also includes blood velocity, viscosity, and pressure but from this complex physiologic process a number of clinical implications are possible; assuming that all other factors are constant, the danger of embolization increases after 10 to 24 hours of fibrillation. In other words, risk of converting paroxysmal episodes of atrial fibrillation is much less than in continuous fibrillation which has lasted for days, weeks, or years.

It has been pointed out that many attacks of paroxysmal atrial fibrillation revert spontaneously after a few moments or within an hour; if the episode does not subside within this period, active measures are indicated. The digitalis drugs by intramuscular injection in the calculated dosage should be given; intravenous medication is not desirable. Slowing the ventricular rate to within normal limits and disappearance of the pulse deficit is a better procedure than the attempt to convert the rapid ventricular rate to sinus rhythm in a short time. Experience has shown that slower rates sometimes convert more promptly than the rapid; certainly the danger of possible embolization is less. If there has been a history of previous episodes, quinidine is indicated after the attack has subsided, provided there are no signs of congestive failure. The suppressive action of quinidine is well established but it cannot take the place of *digitalis* in myocardial failure. While not recommended, the two drugs have been given together in certain selected cases.

Conversion of Atrial Fibrillation

Conversion of atrial fibrillation to normal sinus rhythm is not without certain calculated risks which are dependent upon several known and many unknown factors. The chief hazard is embolization; during a period of atrial fibrillation, the movement of blood within the atria is markedly slowed. Experiments show that the greatest motion is in the central fluid stream and the least at the periphery; where the blood stream touches the atrial wall there may be no movement. In mitral stenosis with marked dilatation and hypertrophy of the left atrium, the internal structure of the chamber with its muscular ridges or columns offer many little depressions where murantic thrombi may develop; the appendage or auricle is a common site for such thrombus formation. During fibrillation there is no coordinated contraction of the atrium; in sinus rhythm, the chamber contracts forcibly as a single entity. During such contractions, fragments of the thrombi or the entire clot may be torn from the muscle wall and, passing through the mitral valve, becomes an embolus in the left ventricle from which it leaves with the blood stream. The brain and anyone of the peripheral arteries may receive the embolus; rarely the coronaries are so involved.

With this factual data in mind, certain observations may be made; the first is in relation to the duration of fibrillation. The longer the abnormal rhythm has dominated the cardiac cycle, the greater is the possibility of thrombus formation. It is estimated that murantic

ent previously. Restoration to sinus rhythm does not necessarily mean automatic return to normal cardiovascular function; there may be little difference in volume output when ventricular rates are in the 60's and 70's, regardless of sinus rhythm or atrial fibrillation.

Chronic Atrial Fibrillation

Chronic atrial fibrillation at the more rapid rates (100 to 140) is most often seen in the mitral stenosis and hyperthyroid groups; as a rule, these patients are in the younger age periods than the slow fibrillators where the basic pathology is chiefly arteriosclerotic and hypertensive disease. Digitalization of the rheumatic valvular group ordinarily presents no great difficulty; various grades of congestive failure are usually present although this may not be obvious. Gain in weight may sometimes be taken as indication of fluid retention when no edema is noted on careful physical examination. Here the diuretics may increase digitalis effect; some of the older authors believe that there might exist a synergistic relationship between the mercurial diuretic drugs and digitalis but the same effect also occurs with the non-mercurial chemicals like Diamox (Lederle) and Diuril (Merck). Simultaneous administration of digitalis and the diuretics frequently lowers the ventricular rate more satisfactorily than digitalis alone, moreover, the dosage of the digitalis may be lessened.

Digitalis ordinarily has little therapeutic effect in atrial fibrillation due to thyrotoxic disease, treatment of the thyroid syndrome will usually be accompanied by some

Procaine amide hydrochloride or Pronestyl (Squibb) has a suppressive effect like quinidine; its action is more prompt and it may be administered intravenously with less reaction than quinidine. Pronestyl will not convert atrial fibrillation to sinus rhythm but it may slow ventricular rate. Where quinidine causes sensitization problems, Pronestyl may be used. Both drugs when taken by mouth over long periods for suppressive purposes may cause gastrointestinal symptoms; quinidine probably produces fewer complaints. Pronestyl, because of its more rapid action, is the drug of choice in the irregularities which occur in the acute coronary syndrome; its routine employment in every case is not indicated but some authors believe it should be administered from the very beginning.

Continuous atrial fibrillation is seen in mitral stenosis, hypertensive heart disease, arteriosclerotic myocardosis, and hyperthyroid syndromes; it may develop and persist in toxic conditions like diphtheria, severe bacterial infections of mixed etiology, and rheumatic fever without obvious valvular involvement. It is doubtful that conversion, even if possible, in these cases serves any useful purpose although some clinicians still recommend at least one attempt; they accept the calculated risk of embolization. Atrial fibrillation, as noted before, is not inconsistent with a long and fruitful life; many slow fibrillators with no pulse deficit are able to perform physical effort with minimal discomfort. They are usually unaware of the irregularity and require little or no medication; conversion to sinus rhythm in some instances has been associated with a number of subjective complaints which were not pres-

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tive failure when there is considerable enlargement of the liver; hepatomegaly may be the predominating clinical finding with minimal edema of the extremities. While the chief site of digitalis metabolism has not been definitely demonstrated, recent isotope studies indicate that the liver becomes the largest storage depot of the drug, with the heart muscle and brain also containing considerable quantities. Failure of the liver to convert the drug into its final form, usable for the heart muscle, apparently is the mechanism responsible for the digitalis-fast condition. Rapid diuresis in such instances may release large quantities of the drug into the circulation with increase in the overdosage symptoms; cautious administration of the diuretics may not only reduce the danger of acute digitalis intoxication but may restore the cardiophilic action of the drug. The non-mercurial diuretics seem to be more satisfactory here, the aminophyllin series may be employed. Choledyl (Nepera) has been useful.

Atrial Flutter

Paroxysms of atrial flutter are usually of short duration; they spontaneously change either to atrial fibrillation or sinus rhythm. Atrial flutter is characterized by rapid complete contractions of the atria; the rate is usually in the range of 260 to 300. Not all the atrial beats are transmitted through the junctional tissue; a functional block develops so that every second, third, or fourth stimulus passes through. Thus a 2 to 1, 3 to 1, or 4 to 1 block occurs producing a ventricular response in the range of 150, 100, or 75 beats per minute. The rapid pulsations of atrial

improvement in the cardiac condition. Restoration of sinus rhythm occurs more rapidly after the use of I^{131} than after surgery of the gland; isotope therapy appears to render the heart more susceptible to digitalization than simple removal of parts of the thyroid. Congestive failure develops in hyperthyroid atrial fibrillaton when the rates are high (160 to 180) and when the pulse deficit is marked (80 to 110).

Atrial fibrillation with congestive failure is sometimes seen in chronic cor pulmonale; digitalization is also accomplished with great difficulty. It may be said here also that diuresis may frequently change the course of the condition and its employment should be considered in all such stubborn cases which do not respond to digitalis alone.

The Digitalis-Fast Syndrome

Some comment should be made in regard to the digitalis-fast syndrome. The digitalis drugs, and this applies to the whole leaf preparation as well as the various glucocides, may be required in the treatment of heart disease over long periods; some patients remain under digitalis control for 30 years or longer. Not infrequently during its employment a time comes when the drug apparently ceases to be therapeutically effective; increasing the dosage may not produce further result. Digitalis intoxication with its characteristic electrocardiographic changes and other symptoms of overdosage may be noted, but the previous favorable effects like reduction of dyspnea may be lost. Such episodes usually occur in conges-

the same hazardous change in hemodynamics as paroxysmal ventricular tachycardia and the development of ventricular flutter and later ventricular fibrillation may occur. There is thus a clinical difference in the prognosis of atrial flutter compared to atrial fibrillation; the latter condition is ordinarily more responsive to suppressive medication than flutter, insofar as maintaining the blocking mechanism is concerned. Quinidine and Pronestyl will have little effect on the junctional area but both may reduce the irritability factors in the atria and ventricles.

Ventricular Tachycardia

From a clinical standpoint, the abnormal rhythms which originate in the ventricles are always more hazardous, both potentially and actually, than all the other irregularities. The potential danger is the eventual termination in ventricular fibrillation; the physiologic steps from nodal tachycardia to extrasystolic ventricular tachycardia to ventricular flutter to ventricular fibrillation may be exceedingly small and may occur very suddenly. The development of any abnormal ventricular rhythm in the acute coronary syndrome carries a guarded prognosis; the outlook is only slightly less sinister when the arrhythmia occurs in other cardiac conditions. Paroxysmal ventricular tachycardia occasionally is seen in "normal" hearts; the normal here is questionable even when no objective signs of pathology can be discovered on careful examination. In this, it differs from the paroxysmal atrial tachycardias which may occur in athletes and individuals with no somatic disease; the ventricular group are more frequent

flutter may sometimes be quite evident in the vessels of the neck; rarely the atrial sounds may be heard on careful ausculation. Radial rates in the 70's may be deceiving; there are many reports of chronic atrial flutter which have gone unrecognized because of the apparent normal rate and rhythm. Exercise in such cases will reveal the abnormality. When the rates are 100 or 150, some irregularity is usually noted and the condition may be suspected from changes which occur in the tonal pattern of the heart sound. Sudden change in ventricular rate from one block phase to another may occur during vagal stimulation; thus a rate of 150 may become 100 or 75. Exercise may also produce this effect for a short interval; the Valsalva procedure frequently produces the change which may be more lasting.

Digitalis is the drug of choice in converting atrial flutter to sinus rhythm; quinidine may convert it to fibrillation and then to sinus rhythm. Pronestyl and digitalis or quinidine in combination have been used in the paroxysmal type, but in persistent or chronic flutter, the digitalis series are more effective. Often, no special treatment is indicated when the ventricular rate is slow; patients may exhibit the syndrome for several years. The author reported a case which lasted for 3 years and Wolffe followed one for 41 months. If congestive failure develops, however, treatment must be started promptly.

Atrial flutter which develops during the acute coronary syndrome may present special problems; in a heart depleted by the anoxial syndrome, the normal blocking mechanism in the junctional area may lose its filtering effectiveness. The rapid ventricular response will produce

suppressive effect of quinidine is useful. Bellet has reported that the drug in the form of quinidine gluconate is long lasting and maintains higher blood plasma levels than quinidine sulphate.

Paroxysmal ventricular tachycardia may occur in complete heart block when this conduction disturbance develops during myocardial infarction; it is uncommon in the chronic types of complete atrial and ventricular dissociation. It is a dangerous arrhythmia and does not respond well to either Pronestyl or quinidine; intravenous magnesium sulphate has been used and some of the atropine group have been employed in the attempt to prevent subsequent ventricular fibrillation. Barium chloride is not usually effective.

Ventricular Fibrillation

Fibrillation of the ventricles is ordinarily a lethal arrhythmia; it is probably the mechanism of sudden death in acute coronary occlusion as a result of functional cardiac arrest. The difference between functional and total cardiac arrest has already been described; resuscitation procedures are more likely to be successful in the latter condition. Irreversible changes occur in the brain after 5 to 7 minutes in cardiac arrest; reactivation of the stopped heart is most effective during the first three minutes. Time is truly of the essence here and prompt measures must be undertaken. Intrapericardial injection of Pronestyl is the first recommended step in resuscitation; if no heart beats are heard after 2 minutes, the heart must be exposed by

in the older and the atrial in the younger age period. It is probably that the development of a diffuse fibrosis of non-specific etiology and of the type seen in myocardosis may be a factor in the occurrence of such ventricular tachycardias in otherwise normal individuals. Autopsy specimens of patients dying during an attack have shown this pathology.

Treatment of the syndrome depends upon the associated cardiovascular status; in the otherwise normal individual, rest, sedation, and vagal procedures may abort an attack. Vagal stimulation is ordinarily far less effective in the ventricular disturbances compared to the favorable results in the atrial group; it is more likely to be successful in the younger normal subjects than in acute coronary disease. Induced nausea and vomiting may stop an attack; sometimes an enema followed by the expelling of flatus is effective. Occasionally heat or cold applied over the precordium has been useful as well as supervised hot or cold baths. In most instances the specific drugs are not indicated since the attack may last only 5 to 20 minutes; it is possible that all of the measures described above are purely coincidental and that the episode may have subsided spontaneously without their employment.

If the attacks persist longer than 20 minutes in the normal individual and promptly when it appears in all other patients, drug therapy is indicated; Pronestyl is the most effective. It may be given intramuscularly and in cardiogenic shock, intravenously with Levophed. In severely damaged hearts with multiple myocardial infarcts, attacks of ventricular tachycardia tend to recur; here, the

suturing of incised tissue should be promptly performed. Attempts to restore the action of the left lung should then be made. Survival is predicated upon speed and attention to the important steps in the procedure; success is gratifying in patients who have literally been snatched from an untimely end.

quick incision below the left diaphragm. The intercostal approach requires more surgical instruments and more time; in cardiac arrest there is no arterial bleeding; if blood spurts from any cut artery a wrong diagnosis has been made. The exposed heart from below is grasped by the physician's both hands and gently but regularly and completely squeezed like a rubber ball. This cannot ordinarily be done with one hand alone and the heart should not be pinched in one area; slow contractions at a rate of 40 per minute are functionally more effective than higher rates. An assistant should carefully palpate the radial artery; unless a beat comes through with each manual contraction of the heart, the procedure is not likely to be successful. The goal of massage is restoration of the coronary circulation and experimental work has shown that a perceptible radial pulsation may be taken as a measure of coronary flow.

Artificial respiration may be required at the same time; collapse of the left lung usually takes place with opening of the diaphragm and care must be exercised not to involve the right diaphragm. There can be no recovery with collapse of both lungs even though cardiac resuscitation has been initially successful. Collapse of one lung in the presence of an already lessened pulmonary functional capacity secondary to congestive failure, requires complete integrity of the right thorax for survival. Manual contraction of the heart is stopped when automatic pulsations occur; the operator will recognize normal or nearly normal contractions of the heart by the characteristic "feel" of its movements; tying of bleeding vessels and

Common Causes of Acute Pulmonary Edema

Statistically, the most common cause of acute pulmonary edema is right sided heart failure; where rheumatic fever is endemic, and this includes the northern band of states across the nation, mitral stenosis is by far, the chief etiological factor. Cor pulmonale due to the various pulmonary diseases may be the important factor in other geographical areas. Both conditions are chronic in their natural medical history and may exist for many years before the onset of pulmonary edema; the patient or his *family can readily answer this question.* Acute pulmonary edema due to the left-sided heart failure is much less common; it may develop in the late stages of hypertension, aortic valvular disease, and in certain types of arteriosclerotic or atheromatous cardiac pathology both with and without coronary artery disease. Here, the question may not be so readily answered but the experienced clinician can usually make a number of correct assumptions. Acute pulmonary edema may occasionally be seen in a variety of acute or subacute cardiac conditions where more or less extensive myocarditis has developed. There are a number or traumatic causes for acute pulmonary edema; inspiration of toxic or corrosive substances or gases may produce a local involvement of the bronchi and lung tissue with overwhelming edema. Diagnosis and hence treatment will be based upon the establishment of the history of these relatively uncommon conditions; they will rarely be seen by the general practitioner compared to the acute pulmonary edema resulting from the chronic types of heart disease.

CHAPTER XI

ACUTE PULMONARY EDEMA

Perhaps the most terrifying and ominous syndrome of all the cardiac emergencies, both from the patient's and family's point of view is acute pulmonary edema.

The restlessness of the patient in acute pulmonary edema is purposeful; it is the ceaseless attempt to assume a better breathing posture. While the diagnosis of acute pulmonary edema can usually be made by simple observation, a brief history of the patient's previous medical status may be important although somewhat difficult to obtain under the emergency conditions attending the situation. The patient as well as the family expect prompt treatment by the practitioner as soon as he arrives and they may well resent any elaborate attempt to obtain a detailed history of the individual's previous illnesses at the expense of extending the patient's discomfort. A few well chosen questions, however, may be made while the physician is preparing for the examination and anticipated treatment. What are these questions? At a Cardiac Conference held at New York City Hospital in 1956 when this problem was considered, consensus approved a minimum of three basic questions: (1) known heart disease, particularly mitral stenosis and/or pulmonary disease, (2) previous episodes of pulmonary edema, (3) if previous episodes, specific response to therapy.

cations it is possible that there is a relationship here. Other measures include the use of drugs which have previously shown a definite preventative or therapeutic effect. The third question to be secured in regard to the medical history of the patient concerns the use of these drugs and the experience gained in the control of previous attacks.

Auscultation

A brief ausculatation of the heart is important and may be very difficult; noisy rales may obscure the heart sounds and the forced respiratory movements may also add to the problem. While rate and rhythm of the cardiac cycle can be determined from the radial artery, the question of murmurs and their identification may play a role in treatment. The presystolic murmur of mitral stenosis or the diastolic murmur of aortic valvular insufficiency, if discovered, will differentiate this type of pulmonary edema from cor pulmonale. Blood pressure may give a clue concerning a possible hypertensive background. Gallop rhythm may suggest a former coronary-myocardial episode. The previously described "one heart sound syndrome," if present, may indicate a serious prognosis. The patient's legs can be quickly examined for more or less extensive edema; correlation studies between chronic peripheral edema and the occurrence of acute pulmonary edema have not been definitive. Diagnosis of hepatomegaly is difficult to establish unless the individual is able to assume a reclining position but in advanced congestive failure a large liver as well as ascites can be determined

Importance of History

The history of a previous episode of acute pulmonary edema *is important; the syndrome is characterized by repeated attacks.* Some patients may experience as many as 20 to 30 episodes over the course of months or years; rarely the first attack is fatal. The episodes may vary in severity and duration; previous reports that each succeeding attack is more severe and more difficult to treat is not borne out by several recent studies. Some patients can anticipate the development of acute pulmonary edema; a variety of subjective symptoms may presage the onset of an attack. A change in respiration, a feeling of heaviness over the chest, difficulty in "taking a deep breath," a slight non-productive cough, a peculiar feeling of suffocation, or the feeling that "there is not enough air in the room" are but a few of the common expressions used to convey the impression of an approaching episode. Of interest in this connection is the claustrophobic concept of Wollheim who has reported the development of this symptom in a group of patients subject to acute pulmonary edema; here the fear of small rooms may suddenly seize the patient, who from previous experience recognizes it as a prodromal sign of an oncoming attack. In this phase, many episodes may be averted or ameliorated by an alert patient; each individual learns sooner or later that certain measures apparently have effect on the development of the syndrome. Some of these procedures seem to be based on sheer fantasy but since acute pulmonary edema may have important psychosomatic impli-

any useful purpose. In the tachycardias usually associated with the syndrome, there are definite contraindications while in the slower rates the accelerator mechanism may be excessively stimulated before the drying effect of the drug becomes therapeutically effective.

Oxygen

Oxygen usually produces prompt relief; the mask method is preferable to the tent or catheter in the initial stages of treatment. Often a few deep breaths of oxygen may change the entire clinical picture. The gratifying response to oxygen in this condition has been compared by many observers to the "blessed effect" of nitroglycerin in agonizing attacks of angina pectoris; both are dramatic illustrations of dynamic pharmacology. Oxygen should be continued for several hours after the episode has subsided; recurrences are not uncommon in certain patients. It should not be administered continuously but in a 10 minute off and on schedule; during the "off period" its further use can be determined. Inhalation of dry, pure oxygen is not without some local reaction in the air passages; coughing may be prolonged or aggravated and hiccoughing sometimes occurs. The latter condition may be difficult to control and may enhance the patient's discomfort.

Phlebotomy

Phlebotomy is indicated in all cases of acute pulmonary edema associated with increased venous pressure; in-

even with the patient in the erect position. It should be emphasized again that there frequently is no relation between evidences of considerable congestive failure with peripheral edema and the development of acute pulmonary edema and it is true that patients who experience the most severe episodes of the syndrome may have little or no signs of other types of congestive failure.

Rate and rhythm of the heart beat deserve close attention; acute pulmonary edema has been seen in complete heart block with a ventricular rate of 20. More often the rate approaches 120 to 160; more rapid rates of 220 to 260 suggest paroxysmal tachycardia. In the latter condition, however, the onset of the pulmonary edema is likely to be less fulminating. Occasionally, acute pulmonary edema occurs in digitalis intoxication with a grossly irregular pulse; if the patient has been on a digitalis routine this should be carefully noted since the drug and its dosage may be a consideration in treatment of the attack itself.

Psychosomatic Factors

All authors agree that treatment of the psychosomatic factors is the first step in control of the situation; Demerol, Dilaudid, Pantopoan, Codeine, should be given hypodermically to lessen the anxiety and restlessness. Morphine may cause additional respiratory problems as well as producing nausea and vomiting in certain responsive individuals. Atropine and a number of belladonna products have been traditionally employed in acute pulmonary edema; it is doubtful that this series of drugs serves

burdens to the struggling heart. The diuretics may be administered with the same precautions given to digitalis, namely, the previous use of the drugs may be a deciding factor in its emergency employment.

A final word about acute pulmonary edema; the condition usually represents a late phase in cardiovascular disease, regardless of the etiological background. It is always a clinical warning that irreversible changes may have taken place and that the patient's entire routine of living must be carefully re-evaluated. Life expectancy is shortened with each attack but the outlook is much improved by scrupulous attention to all of the details which favor a more nearly normal compensation level; these details include not only medication, but also activity both mental and physical, diet, and a number of social and family adjustments; under a well balanced philosophy of living, such patients may enjoy a greatly extended period of happiness and usefulness.

Nocturnal Dyspnea

Nocturnal dyspnea is not uncommon in various types of heart disease; some authors regard it to be an early form of acute pulmonary edema. The mechanism of nocturnal dyspnea is chiefly the result of orthostatic factors; the reclining position tends to pool a much larger volume of blood in the pulmonary vascular system than when the patient is standing erect. The normal gravitational forces which assist in maintaining a competent hemodynamic circuit are lost when the aorta and vena cava are in a horizontal position. Individuals who ex-

creased venous pressure occurs chiefly in backward failure and is seen most often in patients with mitral stenosis, cor pulmonale, and other causes of right sided heart failure. The procedure, to be most effective, requires the rapid withdrawal of 500 cc. of blood, followed perhaps with another 250 to 350 cc. within an hour. A prompt change in ventricular output usually accompanies the drop in venous pressure with lessening of the strain factors of the right ventricle. Removal of blood, however, is predicated upon a normal or nearly normal red cell count; anemia of any type may contraindicate phlebotomy unless later tranfusions are to be considered. Phlebotomy is less useful in acute pulmonary edema due to forward failure with poor output. Here, so-called bloodless phlebotomy with tourniquet closure of the extremities may be helpful in maintaining an adequate circulation in the vital areas previously described by this method.

Intravenous Medication

Intravenous aminophyllin may be required if the above measures are unsuccessful; it should be administered slowly with attention to the secondary reactions which may occur. The digitalis drugs and strophantin group are to be given only after the previous digitalis routine has been studied; if no digitalis has been employed prior to the attack, full dosage may be administered without special problems; when, however, the patient has been on digitalis over a period, careful calculation is required to prevent the overdosage syndrome from adding further

CHAPTER XII

HYPERTENSION

Patients with high blood pressure may be divided into two groups: those with symptoms related to the hypertension and those without. Hypertension is frequently a coincidental discovery; its diagnosis is made entirely from a sphygmanometer reading. The patient has no complaints or objective findings of hypertension which appear to be associated in any way with the basic medical problems which brought him to the doctor. Such patients may show pressures as high as 220/110 with normal cardiovascular blood chemistry and renal studies. More often, the pressures range from 160/95 to 188/100; the individual may or may not be aware of the pressure levels. Should such patients receive the anti-hypertensive drugs?

Asymptomatic Hypertension

Asymptomatic hypertension is frequently a paroxysmal syndrome, there may be daily, weekly, or monthly cycles with return to normal levels in between elevations. The restoration of the lower levels is spontaneous and occurs without medication; correlation factors are unknown in most observed cases. In such patients, it is doubtful that drug therapy serves any useful purpose

perience attacks of nocturnal dyspnea automatically sit up in bed or stand up in order to get a "full breath." In many instances, this simple change in position is sufficient to stop the episode; prevention of the condition requires attention to the physiology involved. Elevation of the upper part of the body is thus important; the conventional types of head rests are useful but many patients complain of insomnia in such a position. Increasing the number of pillows has a number of disadvantages; raising the upper part of the mattress often improves the situation but a hospital bed is perhaps the easiest solution of the problem.

Differential Diagnosis

The margin of clinical differentiation between severe episodes of nocturnal dyspnea and mild cases of acute pulmonary edema may be very small; most patients with acute pulmonary edema have a previous history of nocturnal dyspnea. Treatment of insomnia with large doses of barbiturates or other drugs may be responsible for the development of acute pulmonary edema by lessening or blocking the hypoxial reflexes; semiconscious or unconscious patients may thus pass from nocturnal dyspnea to pulmonary edema because of delayed change in the orthostatic factors. Use of the sleep producing drugs may be a complicating problem here since insomnia is the rule with such individuals.

exhibiting signs of the climacteric in both men and women; hyperthyroidism, hyperadrenalism with and without tumor stimulation, and certain pituitary disturbances are also definitive causes for such hypertension. The renal and toxic types are more complex and require specific and careful management. The organic group like coarctation, adrenal tumors and certain brain neoplasms may call for surgical intervention; high systolic pressure due to aortic valvular insufficiency may occasionally be benefited also by a surgical approach.

Malignant Hypertension

Malignant hypertension has been defined as high blood pressure which does not respond to medication of any kind and which rapidly progresses to an irreversible and terminal stage. Here, the sympatheticotropic drugs or actual sympathectomy is indicated. While the basic pathology responsible for the condition remains unchanged, the secondary effects of the hypertension are reduced and prolongation of life is made possible. Such patients demand exhaustive study and institutional care; the outlook in most instances is not good but continuing research is constantly providing better methods of managing the difficult clinical group.

Self Determination of Blood Pressure

Should patients be instructed in the determination of their own blood pressure? It is now many years since the author posed this question before the New York Cardio-

during the normotensive periods; the alleged suppressive action of certain drugs in individuals with a "tendency" to hypertension is not generally acceptable. Many of these cases sooner or later establish a relatively normal pressure unaided by medical or surgical assistance. There is much *post hoc* reasoning in published data concerning the beneficial effect of this or that drug in this group; perhaps nowhere in clinical medicine is wishful thinking more rampant than in the management of the asymptomatic hypertensive patient with the great number of drugs available at this time. A recent listing showed 163 substances that are recommended.

Symptomatic Hypertension

The symptomatic cases may be divided into 4 major groups; those with predominant anxiety and emotiogenic factors, those with obvious endocrine dysfunction, those with renal and toxic syndromes, and those with organic causes for hypertension. However, in nearly every patient with symptomatic high blood pressure, there is an aura of anxiety neurosis in some degree; until this is removed or reduced to a minimum, other specific measures are usually unsuccessful. Here, the tranquilizers have an acceptable therapeutic place in the management of such hypertensive patients; the physician will use the drug with which he is most familiar and in sufficiently large dosage to strip the syndrome of this factor. In many instances, the tranquilizers will be the only substance required to remove symptoms and restore a more or less normotensive state. The endocrine group are chiefly cases

Bistrium (Squibb), Apresoline (Ciba), Anolysen (Wyeth), Ecolid (Ciba), Methium (Warner), Provell Maleate (Lilly), and Veriloid (Riker) have been satisfactory. Magnesium sulphate and 50% glucose have been employed intravenously. Unless congestive failure and pulmonary edema are present, phlebotomy may do more harm than good; removal of venous blood may cause a reflex elevation of systolic pressure.

A final word about the treatment of the symptomatic hypertensive patient; where over-weight is a part of the clinical picture, diet is mandatory. Salt restriction may be necessary; alcohol is contraindicated. Insomnia is common and should be ameliorated. A routine of rest and exercise is important. Constipation because of diet is frequent and must be corrected. Extremes of hot and cold should be avoided. Some attention should be given to the individual's emotional life and recreational outlook; worry and anxiety are usually constant companions in the hypertensive subject; both can neutralize the beneficial effects of all medication and can nullify the physician's best efforts. Pomeranz once said that one can live with hypertension but it cannot be fought; in few other clinical conditions is peace of mind and tranquility of soul more important.

logic Society; the problem is still debated. Experience has shown that *in the hands of intelligent subjects the procedure* has clinical implications. Daily blood pressure data also serves to establish the diagnosis of paroxysmal hypertension and to indicate the details of the cycle if present. Finally, a daily chart becomes a measure of therapeutic response and permits control of medication. Against these favorable results must be weighed the disadvantages; these are chiefly psychosomatic in origin.

Hypertensive Encephalopathies

The hypertensive encephalopathies may require emergency treatment; the so-called preapoplectic syndrome with its characteristic throbbing headache, vertigo, visual disturbances, flushed face, and pulsating temporal arteries, is a familiar and disturbing clinical picture. The actual *systolic level and the extent of the symptoms may* have no correlationship; patients exhibiting this condition may show pressures from 190/100 to 300/180 mm. Hg. The immediate hazard is cerebral hemorrhage; congestive failure and acute pulmonary edema is the next most common complication, and renal damage is a later problem. In such cases, prompt drop in systolic pressure is mandatory; this may be accomplished by a number of approved *methods. Simple spinal tap* with removal of 5 to 20 cc. of fluid, depending upon its pressure gradient, may result in a rapid drop in systolic pressure. The intraspinal injection of one of the local anesthetic preparations through the same needle may be necessary; the sympatheticotropic and ganglionic blocking are useful. Of these,

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